NOVEMBER, 1951

The Review of Gastroenterology

OFFICIAL PUBLICATION

NATIONAL GASTROENTEROLOGICAL ASSOCIATION

Radiological Diagnosis of Gastrointestinal Tract Disease

Surgical Management of Chronic Duodenal Ulcers

Hemorrhoids: Etiology, Pathology and Treatment

Treatment of Amebiasis

●

Seventeenth Annual Convention
New York, N. Y., 20, 21, 22 October 1952

VOLUME 18 NUMBER 11

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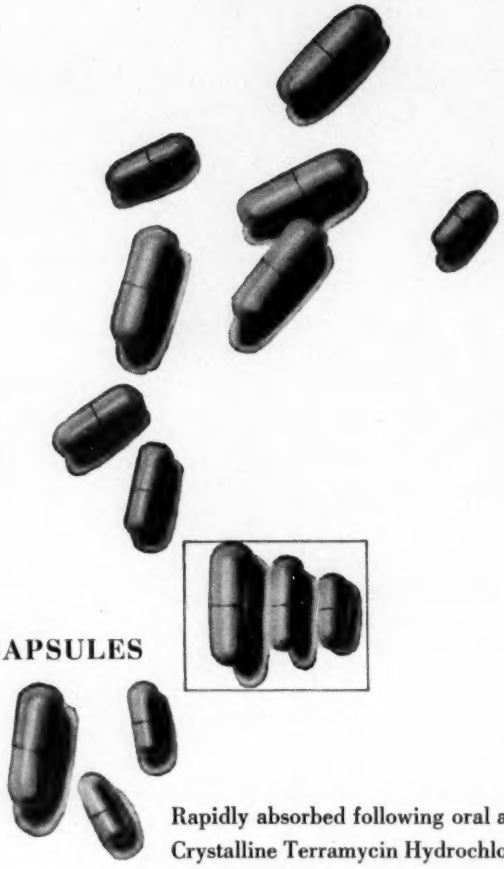

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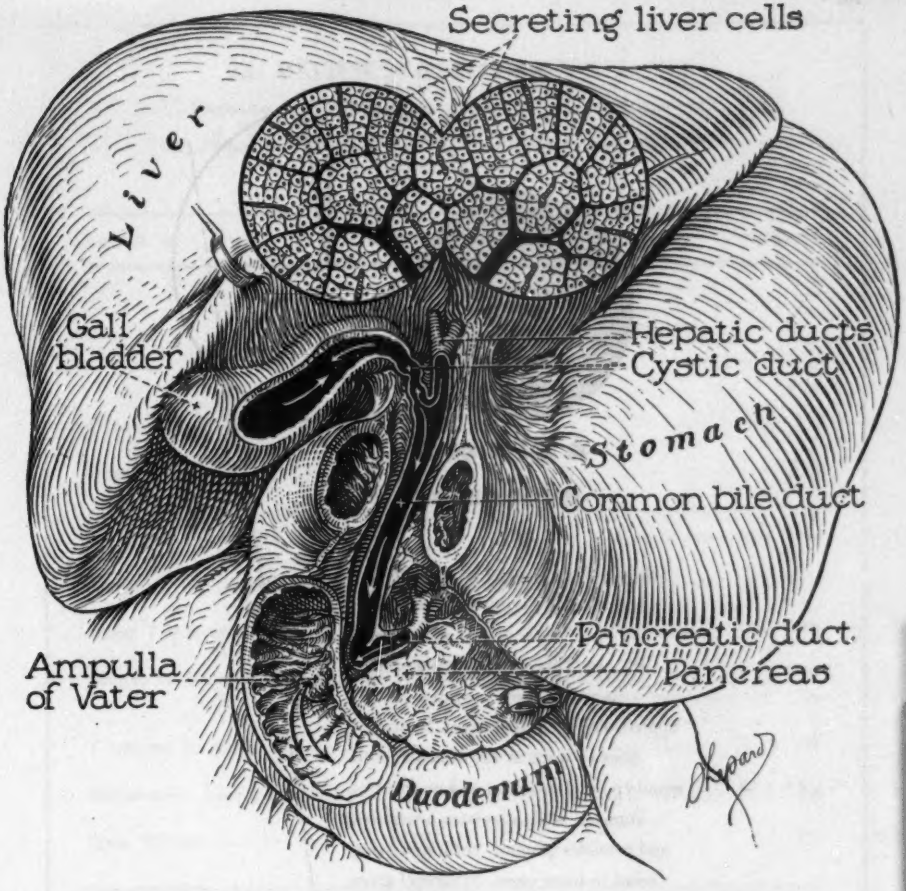

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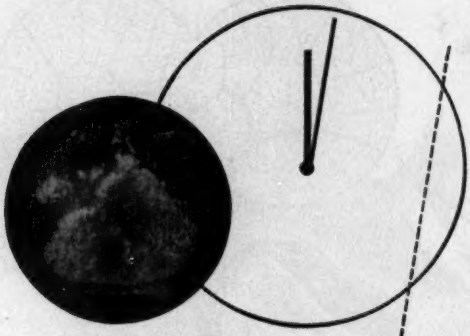

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The Pioneer Journal of Gastroenterology, Proctology and Allied Subjects in the United States and Canada

VOLUME 18 NOVEMBER, 1951 NUMBER 11

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The Review of Gastroenterology

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Volume 18 NOVEMBER, 1951 Number 11

EVALUATION OF THE END RESULTS IN SURGICAL MANAGEMENT OF CHRONIC DUODENAL ULCERS FOR THE PAST TWENTY YEARS*

J. WILLIAM HINTON, M.D.

New York, N. Y.

I want it definitely understood duodenal ulcer is a medical problem and only the complicated ulcer should be considered as a surgical problem.

Unfortunately, in the last five years, with the introduction of vagotomy, many more patients are being operated upon than were ten years ago, not only for vagotomy but for other types of definitive surgery, and I think that we will go back through the same cycle, surgically speaking, in the next decade that we went through perhaps a decade ago, namely, many bad results from too early surgery, regardless of the type of surgery performed.

Twenty years ago, or better, twenty-two years ago, on the Fourth Surgical Division at Bellevue Hospital, I was given the privilege, by the Director of the Service, Dr. Carl Berdick, of starting a clinic to study the end results of surgery for duodenal ulcer. This clinic was manned by internists and surgeons and it was not our intention to corral patients for operation.

In the first five years of that clinic the total number of patients seen is 583 and 143 of those had been previously operated upon; 440 were unoperated patients, and only 33, or 7.5 per cent of those patients were referred for surgery.

It was our feeling at that time that the so-called physiological attack on ulcer, namely, a short-circuiting procedure, would be satisfactory provided the indications were really there for surgery. By referring 7.5 per cent of the patients, it would seem we referred only those patients perhaps needing surgery, but during that same period of time amongst the patients who had been operated upon, some on our service; some in other institutions of New York City whom we had the opportunity of following—we soon found that the percentage of gastrojejunal ulcer was rather appalling, provided you followed the patient long enough. In 7.1 per cent we had 24.5 per cent gastrojejunal ulcers, but the upsetting finding from our point of view was the thirty-three patients we referred to surgery. I had the opportunity of either operating or supervising that surgery.

*Presented before the Course in Postgraduate Gastroenterology of the National Gastroenterological Association, New York, N. Y., 12, 13, 14 October 1950.

I thought that the majority of those patients had anterior-duodenal ulcers, and in that group of patients we had 20 per cent good results with 1.7 years' average follow-up, which is most discouraging.

At that time, late in 1932, we concluded that short-circuiting procedures in our hands brought about an unsatisfactory result. The reason was not clear to me at that time, but in January of 1933 we started doing subtotal resections.

As soon as we had done fifty-seven subtotal resections, it became rather obvious to me why we had failed with gastroenterostomy; namely, we were operating on patients with chronic penetrating posterior ulcers and pancreatitis, where short-circuiting procedure would certainly fail. The duration of the ages of the patients was identical, which would indicate we were operating upon the same type of patient.

From 1933 to 1944 we were very happy. We didn't have any problems.

This summarizes some chronic ulcers, duodenal ulcers, some gastrojejunal ulcers, also operated upon in that period. In 162 cases of chronic ulcer, the mortality (there is a reasonable mortality) was 3.7, and the survivals followed were 135, or 86 per cent, and we had 90 per cent good results. I know those results because I have seen those patients. It is not a letter follow-up; it is a personal interview.

Our achlorhydria on a fasting stomach is only 8 per cent, and after histamine it is 15. In all of our reports, the best operating results in achlorhydria are 46 per cent, and the losses have been about 80 per cent.

It was our feeling that perhaps the major thing in curing these patients was removing the disease process, which is usually the case in most surgical operations. If you remove the pathology, you have a reasonable chance of success. If you leave the pathology behind you, in all probability you will have fewer good results.

The gastrojejunal ulcer is in that group. There were some 27 with a total mortality, of course, of 15 per cent, and 72 per cent good results.

Now, we come to the next era in our lives, 1945 to 1948, and it was a very unhappy era, from my point of view, because I was confused myself. I could not understand why, with the advent of vagotomy, these good results were recurring, and we have three groups of patients:

Subtotal resections without complementary vagotomy.

Subtotals with complementary vagotomy.

Vagotomy as a sole procedure.

In the group without complementary vagotomy, there were sixty-four patients for chronic ulcer, and some four or five for gastrojejunal ulcer. With the complementary vagotomy there were some fifty-six patients with chronic ulcer, and about three or four with gastrojejunal ulcer, and with a total mortality of 1.5 per cent.

The vagotomies amounted to fifty-four for chronic ulcer, and gastrojejunal ulcer; thirty-four or five were for chronic ulcer, and eighteen or nineteen for gastrojejunal ulcer. You will note that our mortality was 9 per cent.

It is certainly an easy operation, and that is the reason so many of them have been done, because it is easy. Some of our complications were of a cardiovascular nature.

The results of these operative procedures, vagotomy operations: The side-effects which we encountered were intermittent diarrhea, flatulence, and cardiospasm, and cardiospasm has occurred in quite a number of the patients with the chronic duodenal ulcers. Thirty-one survived, and there is one patient still totally disabled because of his diarrhea. This patient had worked regularly before his operation, and now, some four or five years later, he is still not working.

There were many partially disabled patients, and, when we sum it up, the satisfactory results in that group of patients were 35 per cent, with an average follow-up of 1.8 years. Of the gastrojejunal ulcers, 18 of the 19 survived. There were four subsequent resections; the good results were 44 per cent, as against 72 per cent without vagotomy—subtotal alone. This 35 per cent vagotomy good results, on the average of 1.8 years, was against 90 per cent good results without vagotomy.

The most distressing thing, from my point of view was the dumping syndrome. I had never had any problem with the dumping syndrome until I added vagotomy, which I thought would be an ideal procedure with a subtotal resection, provided the ulcer was removed. But, we had 24 per cent dumping syndromes in sixty-two patients; 89 per cent were followed on an average of 1.7 years.

Subtotal without vagotomy, during the same period of time, 68.8 per cent, followed an average of two years. The average weight remained the same. There was a weight loss in the other group, averaging up to eighty pounds.

The explanation of why this should occur in the vagotomies: Identically the same operative procedure has been done in the two groups of patients so far as anastomosis was concerned. It is, as I think most people agree, due to rapid distention of the jejunal loops, and apparently there is some suggestive evidence, I might say, that there is some sphincteric control developing in the patient with subtotal resection, and perhaps it does not develop in the patient with an accompanying vagotomy.

Of the 1933 to 1948, inclusive, subtotals without vagotomy, some 257 for chronic ulcer, or gastrojejunal ulcer, 288, 230, (88 per cent), were followed for an average of four and a half years, with only one having a dumping syndrome, and she had a small ulcer but marked hypertrophy of the pyloric muscles. That is the only dumping syndrome in the patients followed, which was 88 per cent of the total operated, and in all of the group of patients, the ulcer has been removed in toto. Three of those patients had a choledochoduodenal fistula, and I think it would appear obvious to any surgeon, if you are going to cure that patient, you must remove that ulcer. The argument is frequently advanced that to remove the ulcer is too hazardous a procedure.

That, I believe, is not a just criticism, because our mortality represents a reasonable mortality. Our residents are doing this surgery, and there were five

other patients with chronic pancreatitis. In this whole group there is evidence that 89 per cent of them had their pancreas involved in this ulcerative process.

The ulcer, as I say, must be removed, in our experience, if one hopes to get the best end results.

The anastomosis, was not changed in the vagotomies, as against those without vagotomy. It is a short-loop, antecolic anastomosis.

Now, from our experience, we have reverted back to the same type of surgical management which antedated vagotomy; namely, that all chronic ulcers needing surgery have a subtotal resection done with removal of the ulcer in toto. Vagotomy is not performed, with one rare exception—and this is a rare exception because, from our experience, you see few of them—that is the young individual who has had two or three massive hemorrhages, who has the x-ray evidence of a very superficial ulcer, or duodenitis, with a very high fasting acidity, of sixty, seventy, or eighty units of free hydrochloric acid on the fasting stomach, recurring; no symptoms of ulcer, then, recurring. I feel that one is justified in doing vagotomy. We have done five or six with two or three good results.

In gastrojejunal ulcer they are not done. We are doing the same surgery, dissociation of anastomosis, removing the ulcer, and hoping we will get as good results now as we did before the use of vagotomy.

Discussion

Dr. O. H. Wangensteen:—We have just listened with interest to this enlightening presentation by Dr. Hinton. He has a vast experience in this field, and in the main, I agree with everything he has said. However, there is one important item in which I do differ with Dr. Hinton markedly, and that is with reference to the necessity of excising the ulcer.

The ulcer is an end effect. I don't think that if an arsonist sets a number of fires, in order to put the fire out, you have to catch the arsonist. I grant you that is a problem, too, in the public interest; but here the concern is to get rid of the ulcer diathesis, and the ulcer will heal if operative causative factors disappear. As a matter of fact, observations have been made on the rate of healing of ulcers. I had occasion to say yesterday that the esophagus, of all the juxtagastric-lying areas, is the most sensitive to injury by acid-peptic juice; yet even a well developed ulcer in the esophagus will heal in 10 to 15 days if one removes the precipitating influence. Unless there is risk of actual or impending hemorrhage, why remove the ulcer? Why not allow nature to heal it in a few days? It is difficult for me to believe that the dumping syndrome in any sense is contingent on whether or not the surgeon removes the ulcer.

I presume surgeons are more alert to this matter of the dumping syndrome than internists are, and I think these cases must be assessed by surgeons themselves. The sincerity of a good relationship between surgeon and patient here is just as important as it is in the cancer problem.

I confess freely that in my patients undergoing gastric resection for ulcer, the incidence of the dumping syndrome, or something we call a dumping syndrome,

is real. John Hunter initiated a discussion on the stomach by saying: "Gentlemen, is the stomach a reservoir or a stewpan?" and he came to the conclusion it was both. This was before we knew there was any hydrochloric acid in the stomach, and in that hypothecation of John Hunter's, of course, we have much of the answer to our own problem.

You take out the stomach for a fellow who has cancer, who already through the years has lost much of the digestive function of his stomach, (because 90 per cent of patients with gastric cancer are either achlorhydric or hypochlorhydric); the stewpan function of the stomach therefore has already been lost in the patient with gastric cancer. In the operation his only additional loss is loss of the reservoir function of his stomach; in consequence, he does not suffer as much as the ulcer patient undergoing gastric resection.

I presume the patient with ulcer becomes very dependent upon his active stomach for the digestion of his food. All at once, he is deprived of his reservoir and stewpan. That is asking a lot of him. Therefore, he suffers a greater loss than the gastric cancer patient. I would say 80 per cent of our patients who undergo resection, have some kind of difficulty in the first four or five months, frequently for six months. Many of them are better at the end of a year. I suppose less than 10 per cent continue to complain beyond that time. My clinic maintains a close relationship with all our ulcer patients who undergo resection. They come back for years and years—some for more than ten years, continuing to visit us once or twice a year. I see them and talk to them. I know them. I know about their families. I would say that perhaps four or five per cent have traces of the dumping syndrome for a long time afterward.

These are not imagined ills, they are real. They say Christian Science is an imaginary treatment for a sure illness, and a sure treatment for an imaginary illness. It is necessary to try to differentiate between what is psychologic and what is physiologic. When a thing occurs frequently, that is physiologic, and it is up to us to try to find out the nature of the disturbance.

The main problem in ulcer is to rid the patient of his ulcer diathesis. How will one do that? Dr. Hinton probably will not remember. I went to see him when he was a Staff man at the Bellevue Hospital in the Summer of 1927, and chased him around and found him finally in the back yard of the hospital. When I saw him next, his hair was gray and mine was white; such is the way we travel through life; sometimes, contacting one another casually and later, more intimately, through the contact of mutually interesting problems. Well, to get back to ridding the patient of his ulcer diathesis: If the internists can do this for ulcer patients, I am all for that; if prayer will do it, I am for that; if a psychiatrist talking to a patient can do it, I am for that—but most of those cures do not hold up—they do not last—and we have to offer patients with ulcer a remedy that cures the ulcer diathesis.

We had the pleasure last evening of hearing Dr. Richard Lewisohn, who introduced gastric resection to this country. When I visited him in the Summer of

1927, he one day did two operations for ulcer: one was a gastroenterostomy, the other an antral resection. When he wrote, years later, in *Surgery, Gynecology and Obstetrics* about the observed incidence of gastrojejunal ulcer following gastrojejunostomy, reporting an incidence of 34 per cent, many surgeons said they saw this complication in only 3 per cent; and others suggested: "Well, they must be operating on an unusual group of patients", or, "They do not do this operation as it should be done, or as we do it". There is not that much difference between surgeons. What happens to you will happen to me, and if we are sincere and honest in what we report, we know that what happens to you will ultimately happen to me, that is, when we perform the same operation. And today, what Lewisohn reported then has been verified in many quarters about the frequency with which gastrojejunal ulcer follows gastrojejunostomy. As Dr. Hinton has said, that operation for ulcer has been thrown overboard by almost everyone. Gastrojejunostomy is an ulcer-provoking operation. Let no one try to resurrect it. Funeral obsequies upon gastrojejunostomy for ulcer already have been held.

Take a dog—he does not have spontaneous peptic ulcer. Ivy and Mann are both authors of that suggestion. Watts, Professor of Surgery, at Charlottesville, Virginia, long years ago, did gastrojejunostomies on dogs, and observed the spontaneous occurrence of a gastrojejunal ulcer following performance of gastrojejunostomy. There is always a lag, of course, between the discovery of a fact and its implementation. And in all our clinics, we need people who bridge the gap between observation and implementation. We need people in the lighthouses scanning the horizon, to watch for significant developments. In 1913, Clairmont and Haberer then still in Vienna, with Eiselsberg suggested: Why not use the Edkins' idea and get out the antral gastrin secreting area of the stomach.

Well, prior thereto, as you all know, Rudolph Heidenhain, a physiologist, at Breslau, had made isolated pouches in the stomach of the dog, and he said the antrum secretes no acid; the acid is secreted in the corpus and fundus. Those were vagotomized Heidenhain pouches. Then Bensley, in Chicago, came along and proved this physiologic observation histologically. You might think that would be easy; it is far from it. We need people of the Bensley type in the United States, and the world needs more Bensleys to study the no-man's land between physiology and microscopic anatomy. Bensley studied the gastric mucosa and found parietal cells by staining them, and thus corroborated Heidenhain's observation. While wishing we had more Bensleys, we probably should be thankful for one.

Then Clairmont and Haberer in 1913, introduced antral resection and presently the small gastric resection became the order of the day, and everybody did them for a while. Presently, in 1918, Finsterer said antral resection was not enough; that it was necessary to go higher on the stomach, and when in 1934, Finsterer wrote another paper upon ulcer, he said it was necessary to remove two-thirds of the stomach to cure ulcer. In 1926 came the suggestion of Willmanns somewhere in Holland, I believe.

Dr. Snapper:—No!

Dr. Wangensteen:—A countryman of yours.

Dr. Snapper:—No.

Dr. Wangensteen:—Willmanns* suggested that, in difficult duodenal ulcer, one should remove the mucosa from the remaining antral fragment removing simultaneously as much corporic and fundic mucosa as Finsterer had suggested in 1918.

I know that keen exponents of gastric surgery like Hinton and Lahey have said you must take out the ulcer. Personally, I do not believe it has anything to do with the ultimate cure rate; and as some of you know, from earlier discussions recently, I have been doing an operation of a different kind. I have reviewed the old sleeve resection, doing it, however, quite differently than Payr did it. I will draw the plan of the procedure on the blackboard. The x-ray people love this operation because now I call them to the operating room and, for the first time in their lives they are seeing duodenal ulcers. Now they can check their x-ray observations by looking into the duodenum at operation.

As Dr. Hinton suggested, we have at our command methods by which we can cure ulcer. I think we can lay the ulcer diathesis and get rid of it. Surgeons have learned how to do it; it has taken a long time. Up until recently, military people, internists, and actuarians have had no confidence in the surgeon's competence with reference to the ulcer problem because the surgeon has been a frank empiricist, trying this and that, not knowing what he should do.

Why did not the sleeve resection work in the hands of Payr? In the first place, he did not take out enough acid-secreting area. I have weighed and drawn out, from Payr's illustrations by the planigraphic methods, Payr's extent of gastric removal. It varied from 25 to 50 per cent, as depicted in his illustrations. I remove from 90 to 95 per cent of the acid-secreting area. The antrum is left and a Horsley or Judd pyloroplasty is performed upon the pylorus to insure satisfactory emptying, in the light of the circumstance that the antral segment is automatically vagotomized by the procedure. Recurrent ulcer has not been observed in these patients. Nor do I have misgivings over failure to cure the ulcer by this means because as I indicated earlier, this operation protects against the histamine-in-beeswax provoked ulcer in dogs. It may be possible by vagotomizing the upper gastric fragment simultaneously, to leave regularly 10 per cent of the acid secreting area without fear of ulcer recurrence.

Some of these patients, however, still have the dumping syndrome. I had hoped that leaving the patient with a minature stomach, might eliminate the stomach syndrome. These patients, however, do not appear to lose as much weight as patients undergoing the typical Billroth I or II gastric resection.

As Dr. Hinton has shown, vagotomy is not a good operation for ulcer. That can be shown experimentally. The stomach no longer empties satisfactorily and a coincidental drainage operation must be performed, which then robs the vagotomy of a good deal of its protection against recurrent ulcer.

*Dr. Snapper was right. Willmanns was at the Gilead hospital in Western Germany.

As Dr. Snapper has said, many times, old things come back. I do not know yet how good the procedure is. I am trying to find out. I have the impression—we have been doing it for about fifteen months and I have done some thirty or forty cases—as I say, I have the impression that this may prove a better operation for ulcer than the conventional operation which is in vogue now.

Frankly, I do not know exactly what this group of symptoms is that one imposes on these patients after the Billroth I or Billroth II operation. The patients are grateful that they can eat many things which they had to avoid before and that no dietary restrictions are imposed upon them; also, that the "Damocles' sword" of ulcer complications has been taken down and put away. It would be nice if we could learn to circumvent these symptoms of quickened gastric emptying too.

I am sure all of you agree with me when I say we have enjoyed very much the big experience reflected in the treatment of this subject by so experienced a surgeon in the gastric field as Dr. Hinton.

Dr. I. Snapper:—When patients with a duodenal ulcer do not heal spontaneously on a bland diet with alkali and belladonna and after giving up smoking and alcohol, medical treatment of duodenal ulcer is about exhausted.

This is the reasons why we physicians listen so carefully when our surgical colleagues discuss their results of subtotal gastrectomy for the treatment of duodenal ulcer. Could perhaps Dr. Hinton tell us what the results are of the simple closure of a perforated ulcer? Which percentage of these patients do well and for how many years does the improvement last?

Question:—May I ask Dr. Wangensteen how he mobilizes the stomach to get a closure after a sleeve resection?

Dr. Wangensteen:—Well, our guest speaker certainly should have the privilege of the last word, so I will speak now. The incision which I showed brings you into the very attic of the abdominal cupola. If you have ever worked inside a cupola, you know how hard it is. We poor surgeons, who must now and then work on big draymen-type of men with paraesophageal hernias know what the problem is; and in latter years, we have gone into the thorax, like a farmer getting out his ladder to climb on top of the cupola from outside it. I feel confident you can get into the attic from the inside by the simple expedient of an extrapleural median sternotomy—that is, by splitting the cupola from below.

I should like to speak to a point, before I sit down, made by Dr. Snapper. Gustav Petren, from Lund, Sweden, wrote in *Surgery, Gynecology and Obstetrics*, in 1913, an interesting article which was a prelude to Balfour's suggestion of the cautery in the surgical management of ulcer. Gustav Petren said, "An ulcer perforates, and the patient is cured." Perforation by no means cures all ulcers. Very few patients were cured by use of the Balfour cautery. It takes more than removal of the ulcer to cure ulcer.

George Thorne, in Boston, has shown, that following an operation, you get an adrenal cortical response, detectable by a lowered esinophile count. I am no

prophet; but I think the time will come when the convulsive therapy of insulin shock employed by psychiatrists will disappear from our medical armamentarium. I hold no brief at all for the surgeon who performs unnecessary operations, but I should think it would do the patient as much good and less harm to have his abdomen opened unnecessarily under anesthesia than to employ some of the shocking methods used by psychiatrists today. Psychiatrists might do well to team up with anesthetists and explore the possibility of better therapeutic relief from anesthetics for some psychiatric conditions. Anesthetics too, provide an adrenocortical response. Psychiatrists use the confused state provided by anesthesia to get information from confused patients—which information in turn often confuses the psychiatrist. Anesthesia in the hands of the psychiatrist might prove to be a better tool than conventional shock therapy. Certainly, it is more humane.

Dr. J. William Hinton:—I am very grateful, Dr. Wangensteen, to you, because you have put this discussion on a level at which I can discuss it.

Dr. Wangensteen is still talking in physiological terms. First I want to get one statement correct. I did not remove the ulcer in toto to prevent dumping syndrome. You see, you misunderstood me on that point. I can see why some of your patients are having trouble. It is very simple, if you look at the drawing Dr. Wangensteen has made.

I have done a reasonable amount of these ulcers and removed them, and in my experience it is impossible for any surgeon to remove all the colloid mucous membrane. If you are going to close the duodenum and leave the ulcer in, you are going to leave in your hormone-secreting mucosa; in other words, you are going to leave in your gastric secretion, or your internal secretion. Of that I am convinced, and when you mobilize your duodenum, whether you close it, use your jejunum, or leave it in and make some anastomosis of the Billroth type anastomosis, you must leave some gastric mucosa at the pylorus because, if you remove that gastric mucosa, you must remove the ulcer, because the ulcer, in our experience, in round figures—90 per cent of them have a posterior ulcer adherent into the duodenum, and to remove the stomach completely, one has to mobilize that duodenal ulcer.

I look upon this problem like cholecystitis and cholelithiasis were looked upon thirty or forty years ago. There were arguments then as to leaving your gallbladder in and taking the stones out. I think that has been forgotten except in the very rare patient who has acute gangrenous gallbladder and is a bad risk, one may remove the stones and leave the gallbladder in.

We know at least in New York, in certain hospitals, they have advocated subtotal resection in massive hemorrhage. I have advocated it, too, since 1937, but in a very minimal percentage of the patients, and in all of the patients we take the ulcer out, because it is a superior pancreatic duodenal artery, or one of its large vessels that has been eroded to.

In one institution in this city they have operated on a relatively high percentage of massive hemorrages, and they have left the ulcer *in situ*. They have

done a subtotal resection, and I think some three of those patients bled to death before they got out of the hospital, from the ulcer.

I know of one or two others who have been operated upon for bleeding ulcer, massive hemorrhage, and have bled to death from the original ulcer, some months, and some weeks, after the original laparotomy, which would in some instances indicate to me that the ulcer has not healed.

I am sure, Dr. Wangensteen, that some of your bad results of these complaints are on a pathological basis, if we get down to earth. I am speaking of a surgeon, looking upon this problem as a pathological one, and forgetting all about the complicated physiology, because we can get confused and will continue to be confused on gastric physiology, and if we can do that, I am sure that the average patient would be better off. That is the only philosophy I have—get your pathology, and, if you get that, you will invariably take care of the physiology, if our concept of the gastrin from the pyloric mucosa is correct, and it is a factor in initiating the acid secretion from the upper portion of the stomach.

Now, of course, Dr. Snapper brought out the perforated ulcer. Those cases, in our experience, have had excellent results, but that is an entirely different pathological process. One has acute perforation in many of these patients. In almost all of those patients they are anterior, simple, uncomplicated ulcer, except of lues, and if you close it, remove the ulcer, 75 per cent will not come to definitive surgery and, as you stated, those patients are cured—but those are not posterior ulcers.

I have had in my experience three patients who have had a perforated anterior ulcer. I had the occasion to operate on one; two others were operated upon in other hospitals, but I had occasion to do definitive surgery in all three. They had a posterior ulcer which was chronic, penetrating into the head of the pancreas and those which had closing anterior ulcer, while convalescing, a week or ten days after the perforation, had a massive hemorrhage in the posterior ulcer, which was uncontrollable by blood replacement, and needed a subsequent subtotal resection.

You can have two ulcers, and in our cases about 2 per cent of the patients have two ulcers, but the patient fortunate enough to have a single anterior operation, for an anterior ulcer, which perforates, if he will use reasonable care, will remain well.

When you talk about putting the patients to sleep, it reminds me of the story that a friend of mine told me here in New York. A lady came in to see him, and she said she had frogs in her stomach, so, after some several months of trying to tell the lady she didn't have frogs in the stomach, she came in and said, "You have got to operate on me because I have a frog in my stomach," so he thought it would be a good idea. He operated upon her and he gave her a frog, so she was perfectly happy for about six months and came back and said, "You have got to operate on me again because you left frog eggs in my stomach."

ALLERGY OF THE GASTROINTESTINAL TRACT*

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It is now generally recognized that of all manifestations of allergy, those in the gastrointestinal tract are the most common. In searching for the cause of gastrointestinal symptoms, allergy must always be borne in mind. It is failure to consider allergy that accounts for so many mistakes in diagnosis and therapy. In my personal experience nearly 20 per cent of patients with gastrointestinal complaints were having some kind of allergic reactions and in 11 per cent of all such patients the principal symptoms were definitely allergic in origin[3,4,5]. Thirty per cent of the latter group had been operated upon, often unnecessarily, as evidenced by the fact that their symptoms had persisted after operation and were promptly relieved when their allergic cause was discovered. Most symptoms for which no organic cause could be determined and which have been attributed to "nervous indigestion", psychiatric and endocrine disturbances, can be demonstrated to be due to allergy. One of the reasons for the present craze for so-called "psychosomatic medicine" is undoubtedly the neglect of careful evaluation of allergic factors.

After the many authoritative reports on gastrointestinal allergy which have appeared in medical literature it is hardly necessary today to discuss the proof of the occurrence of allergic reactions in the gastrointestinal tract. The researches of Walzer and Gray[9,16,17], Rowe[11,12,13], and Vaughan[15] have shown the specificity of these reactions, their rapidity of occurrence after ingestion of allergens, their duration and the effects of therapy. After all, it is the daily experience of clinicians and patients alike to discover that eating of certain foods will invariably produce certain symptoms, such as heartburn, nausea, distention, cramps, diarrheas, migraine, and such directly observable lesions as ulcers in the mouth (canker sores), anus (fissures, pruritus), and in the rectum and sigmoid (ulcerative colitis). One outstanding feature of ordinary allergic reactions is their rapid disappearance upon withdrawal of the offending factor, as seen in urticaria in the skin and through the proctoscope in the lesions of an uncomplicated ulcerative colitis. Frequent repetition of such reactions, however, may result in more or less serious chronic organic changes, which may become intractable or complicated and require operations or may even result in death.

The reason for the occurrence of allergic reactions in general is not known. It is a well-known fact that they often occur in otherwise normal individuals, although they frequently follow acute infections or operations or result from chronic diseased conditions. In the individual there may be a seasonal variation in sensitivity, a variation in the regions in which reactions occur and a variation in the substances to which the individual may be sensitized. Where seasonal variations occur they may be found to be due to sensitivity to pollens, emanations or dusts occurring at certain seasons, or in the cases of milk allergy, to variations in

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the diet of the cows from which the milk was obtained. Variations in location of allergic reactions may be an aftermath of disease, as in cases in which hay fever or asthma may follow an acute respiratory infection, eczema an acute skin infection or ulcerative colitis a specific enterocolitis such as bacillary or amebic dysentery or even an acute salmonella or staphyloccocus infection. An acute febrile reaction due to any cause, such as infection or operation, or foreign protein injection, may result in temporary or permanent cessation of allergic manifestations or may be followed by exacerbations or new manifestations. The reason for the variation in the substances to which the individual is sensitized is not known, but it has been suggested that over-indulgence in some particular food, or over-exposure to some other external factor may play a part in the production of sensitivity, and long continued avoidance of such substances may gradually cause a cessation.

Localization of Manifestations

As allergic reactions may occur in any part of the body, so in the gastrointestinal tract they may be found from mouth to anus and may occur in one localized area or throughout the entire tract at one time. Involvement of the entire tract presents the picture of an acute gastroenteritis, with vomiting, cramps and diarrhea, lasting as long as the offending allergen is being taken or remains in the gastrointestinal tract and often continuing for a few days thereafter. Chronic manifestations almost never occur throughout the entire tract, although both ileum and colon are frequently the site of reactions occurring in such close succession as to be practically continuous, and will produce the picture of an ulcerative colitis.

Usually the manifestations are more or less localized. In the *mouth,* stomatitis, canker sores, glossitis and angioneurotic edema are most frequently seen and cause the characteristic symptoms of these conditions. In the *esophagus,* similar edema, redness, ulcerations or infiltration may cause symptoms suggestive of spasm or of organic obstruction. I saw a patient once who presented the picture of an esophageal carcinoma caused by sensitivity to chewing tobacco. In the *stomach* and *duodenum* the symptoms may be only those of reflex irritation and retrostalsis, may resemble those of gastritis or may be typical of ulcer. In fact occasionally real allergic ulcers may be encountered. In the *small intestine,* mild irritation may produce pains and diarrhea, more chronic changes may produce the typical findings of regional enteritis. The *gallbladder* and *appendix* have been shown to be the occasional site of allergic reactions, with symptoms pointing to inflammation in these organs, often subsiding quickly after epinephrin or an antihistaminic. The *colon* is frequently affected. Mild allergic reactions may be accompanied by peristaltic unrest, mild tenesmus, or diarrhea, often alternating with periods of constipation, and mucus may be seen with formed or loose stools, producing the syndrome known as mucous colitis, irritable or unstable colon. Chronic irritation, due to constant ingestion of the allergen, may cause changes in the bowel wall starting with redness and edema and going through the stages of areas of ischemia, sloughing, hemorrhage, perforations, adhesions and obstructions, producing the condition known as idiopathic ulcerative colitis or colitis gravis[2,6,8,14]. Occasionally, with

symptoms of an acute intestinal obstruction or an "acute abdomen", a surgeon may operate and either find no lesion at all or may find a generalized edema of the colon, which often subsides as he handles it[4]. Such an attack can often be spectacularly relieved by a single injection of epinephrin. In the *anorectal region* pruritus ani is the most common allergic manifestation and may be accompanied by ulcers or fissures, cryptitis or papillitis, often with bleeding.

Diagnosis

Establishing a diagnosis of gastrointestinal allergy is not a simple matter, and few allergists or internists seem to care to take the time to conduct a detailed study such as is necessary. The variations in symptoms and signs, depending upon the location of the local allergic reaction, upon its severity and upon the frequency of its occurrence, cause confusion in diagnosis. They often resemble those of true organic disease, so that it is necessary in every case, before considering the diagnosis of allergy, to rule out any possibility of organic disease by a complete gastrointestinal study and, if indicated by the symptoms, also complete studies of other systems, such as the cardiovascular system, the respiratory tract, the genitourinary tract, and the nervous system. Frequently I see patients who have been treated for allergy in a haphazard fashion when they really have had organic lesions, particularly gastrointestinal carcinoma, now inoperable. On the other hand, it may be postulated that repeated or chronic irritation produced by allergic reactions might be a factor in the production of cancer. In a series of patients with gastrointestinal carcinoma, I found the incidence of previous definite allergic manifestations to be more than double that usually found with other gastrointestinal lesions[7].

A careful *history* will often give valuable information. The significant findings in the history may be classified as follows:

1. *Family history*:—The tendency to allergic reactions being a dominant hereditary factor, makes important a history of such reactions in a family. The manifestations in parents or collateral members of the family may not have been manifested at all in the gastrointestinal tract. A mother with hay fever, or a father with migraine may have a child with only gastrointestinal manifestations.

2. *Previous history*:—Although some patients exhibit only gastrointestinal manifestations, many give a history of previous or coincident manifestations elsewhere, such as hay fever, asthma, migraine[1] and skin or urinary reactions. A history of known reactions following ingestion of certain foods is of value. The patient who says he has heartburn after eating oranges or chocolates, nausea or vomiting after eggs, migraine after whiskey, or who says that milk has always caused diarrhea, should be suspected of allergy. Other unexplainable symptoms, sometimes occurring on certain days of the week, as on Friday nights in persons eating fish only on that day, may be occasioned by allergy. The relation between allergy and endocrine imbalance is often exemplified by the onset or disappearance of symptoms at puberty, during pregnancy or at the menopause. The occurrence of allergy after acute infections must be remembered when patients state that their

symptoms date back to an acute food poisoning or dysentery. Intestinal parasites may cause allergic reactions. As a result of repeated or chronic symptoms patients may develop character changes or neuropsychiatric symptoms, many complain of anorexia, with resulting decrease in food intake, loss of weight and manifestations of dietary deficiencies. Fever may occur from allergic reactions alone or may be the result of secondary infections or the absorption of toxic materials from areas of ulceration or sloughing.

Roentgenologic examination is important, not only to determine the presence of any organic lesion but to demonstrate certain changes in gastrointestinal function and form, quite characteristic of allergy. Motility changes include spasms, mucosal irritability, writhing peristalsis with retrostalsis seen particularly in the duodenum, and irritable or unstable colon. These and mucosal changes, such as puckering, coarsening, or obliteration of mucosal markings, fuzziness, smoothness or defects in the colonic outlines, may be particularly evidenced when the opaque meal contains flavors or foods to which the patient is sensitized, such as milk or chocolate with a barium meal or the iodine in the chemicals used for cholecystography. The chronic infiltrative changes, adhesions or perforations, can of course be demonstrated.

Endoscopy of esophagus, stomach and rectosigmoid, besides helping to rule out organic disease, may demonstrate the redness, edema, infiltration or ulceration associated with allergic reactions and may be used to show improvement after withdrawal of known allergens and recurrence upon their resumption.

A *physiological test* of value particularly in some sudden acute reactions suggesting an "acute abdomen" is the parenteral injection or even sublingual administration of epinephrin, ephedrine, pituitrin or one of the newer antihistaminic drugs, when rapid subsidence of symptoms and signs will suggest allergy as a cause of the acute attack.

Laboratory examinations should not be neglected, not only to rule out organic disease but to strengthen the diagnosis of allergy. The finding of gastric or pancreatic achylia, which may facilitate the absorption of unchanged protein, may be of value, although it is now known that unchanged protein can be absorbed through the normal gastrointestinal mucosa. Eosinophilia or the finding of excessive numbers of eosinophiles in the stools may be of great importance if parasitic infestation can be ruled out. Leucocytosis or an increased sedimentation rate, while it may occur in simple allergic reactions, should cause a suspicion of secondary infection. In severe cases, with anorexia, vomiting, diarrhea or bleeding, evidences of secondary anemia, hypoproteinemia, chemical imbalance and dehydration, must be determined by suitable laboratory studies.

The *findings of the specific allergic factor* is the real problem in any disease due to allergy. In hay fever, asthma and some other allergic manifestations, the familiar skin tests have long been successfully used to determine sensitivity to inhalants such as pollens, grasses, emanations and cosmetics, but it is now generally agreed that such tests for food are of little value, often merely confusing the

TABLE I
DIARY STUDY FOR ALLERGY

	Diet	Unusual Events	Symptoms	Bowels
Oct. 9 8 a.m.	Orange juice *Oatmeal, cream White bread, butter Coffee, cream	none	10 a.m. Heartburn for 1 hr.	8:30 formed
12:00	Steak, *onions Peas, carrots White roll, butter Coffee, cream Stewed peaches		3 p.m. Cramps, off and on for 2 hrs.	3:00 loose
3:00	Vanilla soda, ice cream			5:00 loose
6:00	Lamb, potatoes Carrots, spinach Lettuce, tomato White bread, butter Rice pudding			
Oct. 10 8 a.m.	Orange juice Cornflakes, milk Egg and bacon White roll, butter Coffee, cream	baby sick	nervous all day	7:30 soft formed
12:00	Frankfurter, roll Cole slaw, mustard Vanilla ice cream			
3:30	Coke, pound cake			
6:00	Veal cutlet, potato Broccoli, peas White bread, butter Stewed peaches Tea, lemon			
10:00	Ginger ale, vanilla crackers			
Oct. 11 8a.m.	*Prune juice Cream of wheat, cream Egg Corn bread, butter Coffee, cream	none	9 a.m. heartburn for ½ hr.	8:30 formed
12:00	Roast beef, white bread Coffee, cream *Apple pie		3 p.m. cramps 1 hr.	
4 00	Rye whiskey, soda			
6:30	Bluefish Cole slaw Potatoes Stewed peaches Tea, lemon			

Suspected:—Oats, prunes—heartburn
Onions —diarrhea
Apple —cramps

issue. Most clinical reports disparaging to the theory of food allergy as a cause of gastrointestinal manifestations have used skin tests as the only criterion of allergy. The findings of the food or foods responsible for reactions in the gastrointestinal tract is an arduous task. It requires infinite patience on the part of the physician, patients and attendants, and the most careful attention to detail. Occasionally a history of known sensitivity to a specific food may be of great help. A patient with ulcerative colitis, who has long known that milk caused diarrhea and who has avoided it except as an adjuvant to cereals, beverages or desserts, and who has been worse on a milk and cereal diet prescribed for him by his doctor, can often be spectacularly cured merely by the entire elimination of milk from his diet. Unfortunately however, these cases are few and far between. It is also unusual to find a patient who is sensitive to only one food, although one may be the principal cause of trouble, and its discovery and elimination may cause a marked improvement in symptoms. In general it must be borne in mind that symptoms occurring at intervals and not lasting longer than a few hours or days must be caused by foods taken only at such intervals, whereas symptoms occurring daily and becoming progressively more severe must be due to foods taken with great frequency, several times a day in severe cases such as ulcerative colitis.

Diet study is the most reliable method of determining specific food sensitivity. Various forms of study have been recommended. *Diary study* is applicable in patients with intermittent attacks and consists of keeping an accurate on the spot record of all foods ingested, including all flavorings, condiments and adjuvants, arranged in a vertical column along side of other columns recording time of occurrence and character of symptoms, bowel movements and any other pertinent factors, (as shown in Table I).

By varying the daily menus, by carefully studying the relationship between certain foods and the onset of symptoms, and by keeping three lists, one of foods proven to agree as a result of absence of symptoms following their ingestion, one of foods suspected of causing symptoms and a third of foods found to cause definite symptoms on two or three separate occasions, a satisfactory determination of offending foods can be arrived at in three or four weeks.

Elimination diets, as worked out in detail by such experts as Rowe[12,13] and Vaughan[15] have been found to be surprisingly accurate. However, they require such attention to detail, such intense cooperation on the part of the patient, and usually so much time and frequent consultation between the patient and either his doctor or a dietician expert in this field, that they are not ordinarily practicable. In general they are of value principally in allergy of the intermittent type.

Addition diets can be used in either the constant or intermittent type. The principle of this method of study is first to induce, by dietary methods alone, remission or entire cessation of symptoms. This can often be accomplished by a period of fasting, allowing only water, by the use of only one food, like rice with water (the old Duncan-Bulkeley diet) or by the ingestion of only a few foods for a period of three or four days. Upon subsidence of symptoms, one or two new

foods are added to the diet each day so as to build up rapidly an adequate diet while observing carefully the effect of the new additions. Foods suspected of causing reactions must be tried out two or three times to prove their harmfulness and entirely rejected from the diet when so proven. In most cases, especially in patients who are not in physical condition to permit fasting, it is possible to work out a simple preliminary diet of foods which are not to be suspected of causing symptoms and with elimination of symptoms on this diet, in a few days to begin with additions to it. For many years I have been employing two simple test diets, each consisting of only 5 or 6 foods, which I have identified as test diets, #1 and #2, as follows:

Diet #1, is used in cases in which the patient has been having intermittent attacks of symptoms suspected of being caused by food sensitivity, with no symptoms between attacks, indicating that the food or foods causing the attacks are taken only intermittently. Obviously, the test diet for such a patient should consist of foods ordinarily eaten every day by the average individual. In case the patient suspects one of these common foods and has been avoiding them, such food should of course be eliminated from this diet, which is usually as follows:

Allergy Test Diet #1

5 *Foods*:—Milk, Egg, Wheat, Potato, Orange

Breakfast:—Milk, egg, wheat cereal and cream, wheat bread and butter and orange

Lunch:—Milk, egg or cheese, potato, wheat bread and butter, dessert (orange flavored custard or wheat bread pudding or whole orange)

Supper:—Same as lunch or breakfast

Between meals and at bedtime:—Milk with cream and glucose and wheat crackers

In the absence of symptoms this well-balanced diet can be followed for a long time and while following it the patient can prove the harmfulness of other foods by trying them repeatedly and noting the effects. By laying out a definite program of additions, offending foods can be fairly rapidly discovered. If symptoms are produced or aggravated upon the institution of this test diet, it indicates that at least one of its constituents is at fault, and this must be determined by elimination of one of the five foods at a time or by the use of Diet #2, which consists of foods not ordinarily eaten every day by the patient, thus usually avoiding all of the foods in Diet #1. It is particularly applicable to cases presenting daily or continuous symptoms, such as heartburn, abdominal pains, persistent diarrheas and ulcerative colitis. Even in a severe ulcerative colitis a definite change can be noted within 2 or 3 days after the food or foods to which a patient is allergic have been eliminated from the diet, not only by noting a decrease in tenesmus and in the number of stools, a slightly less liquid consistency of the stools, and less blood, but by improvement in the general appearance of the patient. This diet is of necessity not well-balanced, and must be followed to the minutest detail to avoid too long a

period of food deficiency in an already depleted patient. Any clandestine additions to this diet must be avoided. I once saw a patient with a most severe ulcerative colitis who was not helped by the diet because he was taking orange candy when orange was the principal cause of his disease. The diet is as follows:

ALLERGY TEST DIET #2

5 Foods:—Gelatin, 2 cereals, 1 or 2 vegetables and 1 fruit

N.B. The cereals, vegetables and fruit are chosen after detailed consultation with the patient seems to indicate that they are probably not responsible for symptoms. Rice, rye, peas, carrots and raspberries or grapes are particularly desirable and easily taken and are used below as an example.

Breakfast:—Rice, boiled or puffed, Ry-Krisp, raspberries, gelatin drink

Lunch:—Boiled rice, peas, carrots, Ry-Krisp, gelatin drink, raspberries or raspberry jello

Supper:—Same as lunch or breakfast

Between meals and at bedtime:—Gelatin drink, Ry-Krisp and raspberry jam.

The gelatin is added as a protein because I have never observed a patient who was sensitive to gelatin. The "drink" is made by dissolving a teaspoonful of plain powdered gelatin in hot water and diluting this with water and if desired the juice of the berry or fruit selected, or by adding the gelatin directly to the juice and stirring it in as a powder.

During the use of this diet, a badly debilitated patient may be helped by the use of transfusions, although reactions to foods eaten by the donor may cause reactions. The same may be said of the use of parenteral protein feedings. I prefer not to use either during the 2 or 3 days of the preliminary study and to consider the infusions when used, as additions to the diet.

If symptoms are not promptly alleviated, changes will have to be made to this diet as indicated by careful study of the reactions in relation to items in the diet. As soon as symptoms appear to be relieved, preferably after a day or two, once daily additions are begun and should consist first of proteins, usually meat and later of vegetables, fruits and cereals, bringing the patient as rapidly as possible up to a maintenance diet. The common foods of Diet #1 should be avoided until a nutritious diet has been attained without them, as a severe reaction following their use may delay for days the patient's progress toward proper nutrition.

After certain foods have been proved to be the cause of symptoms the patient should be warned against trying them out for some time to come and should be provided with a well-balanced diet containing foods which have been proved to do him no harm. It must also be explained that as time goes on new sensitivities may occur and old ones may be lost, so that the lists of foods agreeing and disagreeing will of necessity vary somewhat from year to year.

Other factors than food may cause gastrointestinal symptoms and must be determined. Sensitivity to inhalants may not only produce gastrointestinal symptoms in connection with their effect upon the respiratory tract, but in some cases the gastrointestinal tract may be the only shock organ. Attacks of diarrhea and

even ulcerative colitis occurring during the periods when hay fever is common, may be due to pollen allergy. Sensitivity to some chemical constituent of "smog" or in a dusting powder may cause definite gastrointestinal symptoms. Some individuals are sensitive to certain odors. I have a patient who gets an attack of vertigo and vomiting after smelling a certain perfume. Allergic reactions may also result from absorption of cosmetics or medications applied to nose, lips, mouth, anal region or other orifices and perhaps even to the unbroken skin. I have seen cases of persistent vomiting from some particular brand of lipstick or dentifrice. Many patients are found to be allergic to some constituents of the medicines which have been prescribed for them, so that it is important to avoid all medication during a study for allergy. Sensitivity to vitamins is usually not specific for the vitamin but is due to the sensitivity to the source of the vitamin, as the fish from which the liver oil is extracted or the cereal, usually wheat, the germ of which is the source of the vitamins. Some patients may even be sensitive to the drugs used for the relief or prevention of an allergic reaction, such as endocrine preparations or antihistaminics. In some cases such sensitivities can be determined by skin tests, in most cases they must be established by trial as in the case of foods. Taking all of these factors into consideration makes the job of discovering the cause of an allergic reaction no easy chore.

Treatment

Aside from the knowledge that allergy is hereditary, nothing is yet known as to its cause. Therefore there is no specific treatment for this condition except the avoidance of the food or foods or other factors which are found to cause reactions. Theoretically when all such factors have been eliminated the patient should be entirely relieved of symptoms and this is true if no complications have occurred. Fibrotic or polypoid changes caused by long-continued insults, walled-off perforations, scars or adhesions, may show very slow improvement or may cause persistent symptoms which may require operation. The stiffened colon resulting from long continued severe ulcerative colitis may be the cause of a slight diarrhea for months or years, but will usually show gradual improvement as time goes on. Exacerbations may be due to the ingestion of some factor known to be the cause of symptoms or to the development of sensitivity to some new factor. The patient must be instructed always to be on the alert for such occurrences and to determine the cause at once.

The diet, after elimination of all foods to which the patient is sensitized, should be well-balanced, nutritious and agreeable to the patient. There is no general contraindication to roughage (or "smoothage" as it is now called) even in diarrheal conditions. Our Allergy Diet #2, with all its roughage, will cause rapid subsidence of diarrhea even in severe ulcerative colitis.

Medication is usually only palliative, and must be used with great caution because of the good possibility that the patient may be sensitive to drugs as well as foods. As already mentioned, epinephrin or ephedrine are sometimes of great value in causing subsidence of acute lesions such as "angioneurotic" edema of the

bowel, but they are of no value in chronic allergic states. In fact their thyroid stimulation may cause harmful side-effects. Other hormones, such as pituitary and parathyroid, are in the same category. Calcium is at times of considerable value. Antihistaminics may be of considerable value in some cases, but on the whole are not of much use in food allergies. At times racemic amphetamine (benzedrine) will relieve acute gastrointestinal symptoms. Sedatives, antispasmodics, hematinics and vitamin preparations may be used as indicated. Immunologic and antibiotic treatments may be necessary for complications. All medication, even the antihistaminics, must be tried out carefully to rule out sensitivity to them.

Hygienic care is of course indicated. Rest, sunshine, fresh air, recreation, psychological care, attention to any organic disease conditions and eradication of all focal infections are most important.

Desensitization to foods causing symptoms sounds logical in view of the success obtained by this method in the prevention of hay fever and other allergic manifestations by the injection of progressively larger amounts of pollen or other extracts. It must, however, be realized that to maintain desensitization to any substance the body must be exposed to this substance frequently, practically even daily, or sensitization will recur. In hay fever this can be accomplished by having the desensitized patient inhale pollen by keeping a cheesecloth bag of it in the bedroom during the intervals between the seasons when it is found in the air. In the case of food, therefore, the only foods to which the patient should be desensitized should be those ordinarily taken every day and which it is difficult to eliminate entirely from the diet in ordinary daily life. Milk, egg and wheat are really the only foods in this category. In pollen desensitization, owing to the minute quantities to which a patient is normally exposed, the hypodermic injection of dilute extracts in increasing dosage is necessary. In the case of foods, such minute doses are rarely necessary and desensitization can be carried out by the ingestion of gradually increasing amounts of the foods. Rarely can a patient be made to reach a point where he can partake indiscriminately of the offending foods, but he may be able to take measured quantities with no untoward effects. Milk can be used in doses of one drop the first day, increasing the increment as doses get larger. In the case of eggs, a solution of one drop of beaten egg in 250 c.c. of water can be used in a similar manner, and in the case of wheat, small pellets of wheat bread can be made by the patient and the dose increased daily. Nonspecific desensitization by artificial pyrexia is not usually of permanent value and the use of increasing doses of specific food proteoses, peptones, amino acids and histamine have been found to be of little or no value.

The *treatment of an acute attack,* of an allergic reaction accompanied by an acute diarrhea or by symptoms resembling an "acute abdomen" or an acute intestinal obstruction, requires care and discretion. The patient may really have an acute abdomen, so every means of diagnosis must be used to evaluate the situation. In addition to a careful physical examination some laboratory measures should be employed. Scout films of the abdomen may show free air in the peritoneal cavity

in the presence of perforation or the distribution of gas in the intestine may show the site of an obstruction. An elevated sedimentation rate may indicate an acute infection or a severe chronic one. A leucocytosis may occur either in infection or allergy, but if there is a predominance of eosinophiles, allergy is suggested. Fever and pulse rate are more liable to be elevated in the presence of infection. It is practically always safe to give an intramuscular injection of epinephrine (.7 to 1 c.c. of 1 to 1,000 sol.) even while preparations are being made for operation, and in allergy cases the results are startling. A patient with all symptoms indicating the need for immediate operation may, within 10 or 15 minutes, become perfectly comfortable, with complete disappearance of the symptoms and signs. After this, a complete study is of course indicated. It is surprising how many appendices removed for acute appendicitis, show extensive infiltration with eosinophiles, suggesting that possibly allergy was the cause of the acute condition.

Summary

1. Allergic reactions are very common in the gastrointestinal tract.

2. These reactions may result from sensitization to foods, pollens or other inhalants, chemicals and drugs.

3. The reactions may be produced by local application to the mucosa or by offending factors in the blood which have been absorbed after ingestion, inhalation or perhaps even application to the skin, especially if abraded.

4. The symptoms produced by allergic reactions vary with the location, size, depth and durations of the reactions and with the complications produced by them.

5. The symptoms may simulate those of organic or infectious disease or may be bizarre and unexplainable on an organic basis.

6. A diagnosis of gastrointestinal allergy should not be made until a complete gastrointestinal study has been made to rule out organic disease.

7. Roentgenological and endoscopic examinations may help in the diagnosis of allergy and may be used to check results of treatment.

8. Finding of the allergic factor is accomplished by means of a careful history and by diet studies.

9. The use of endocrine, vasoconstrictor or antihistaminic drugs may be of temporary value and desensitization may help, but treatment consists essentially of complete avoidance of all allergens.

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Discussion

Dr. I. Snapper:—In general terms certainly everyone will agree with Dr. Andresen that allergic manifestations in the gastrointestinal tract do occur. Everybody knows of urticaria and gastrointestinal disturbances after strawberries, seafood, aspirin, phenobarbital, etc. These allergies are certainly not completely innocent. I have seen patients who continued taking phenobarbital although they developed hives after this medication, and who ultimately developed thrombopenic purpura.

Whether it is permitted to take another step and surmise that ulcerative colitis is frequently due to allergy, is another thing. An allergic reaction superimposed upon a colitis will certainly make the inflammation worse. Thus one must go along with Dr. Andresen that for treatment of allergic and ulcerative colitis, requires the exclusion of all allergens. Whether this proves that an ulcerative colitis is due to allergy, remains to be seen.

Dr. Albert F. R. Andresen:—In closing, I wish only to discuss one subject, that of ulcerative colitis. I am constantly seeing patients who previously have been subjected to all of the known treatments for ulcerative colitis, including typhoid vaccine intravenously, immunization to specific organisms, psychotherapy, and thorough antibiotic therapy, and they still have ulcerative colitis. Then on diet alone, without any medication, these patients promptly begin to get well. Following up these patients over the years, we find exacerbations if they eat the foods to which they were allergic or new ones to which they have become allergic, but otherwise they remain well for years, with even gradual disappearance of polypoid lesions. If that is not proof of an allergic etiology I do not know what would be considered proof.

NEWER TRENDS AND TECHNICS IN GASTROSCOPY*

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During a decade and a half of development of gastroscopy with flexible instruments it has firmly established itself as a diagnostic procedure. The place for gastroscopy is not that of a competitive method with roentgenography but rather that of an important supplement in the diagnosis of gastric disease. We have come a long way through a period of indecision and reluctance. Gastroscopy can now be said to have definitely arrived and to have received wide spread acceptance. It has demonstrated its value based upon the accumulated experience of a faithful group of workers, among gastroenterologists in America and throughout the world who have made serious study of this diagnostic method.

Newer Developments in Technic:—A study of the accumulated experience of gastroscopists indicate that probably the most vulnerable aspect of gastroscopy today is the existence of blind spots. They are three areas of the stomach, in order of importance, as follows: Small portions of (a) the lesser curvature of the antrum, (b) of the posterior wall and (c) of the fundic or cardiac area. These blind spots exist not in every case but in the great majority of cases. This is a technical or mechanical problem. During the last decade there have been some advances in the direction of solving these important problems. The concerted experiences of gastroscopists indicate that about one-third of the errors in diagnosis are due to inability to see the lesion for technical or mechanical reasons. If the lesion can be seen it can be correctly diagnosed in the great majority of instances. Coating of lenses and improvement of lighting have reduced to some extent the technical problem. Instruments with a wider angle of vision such as the Cameron Omniangle instrument which does not sacrifice magnification are available. Another development is the Eder-Chamberlain movable tip gastroscope or the Herman Taylor English model. These instruments have a manual lever capable of making the tip bend back and forth so as to push away the gastric wall which tends to bring blind areas into view[1]. Several years ago we published an article in THE REVIEW OF GASTROENTEROLOGY recommending the "dextralateral" or right side position in cases of situs inversus and in visualizing "blind spots". From these earlier observations we have developed the "multiple position" or "rotation" technic which is now routine in our clinic at the Cook County Hospital. Patients are first examined in the standard left side position then carefully rotated to the dorsal and when necessary the patient is further rotated to the right side, without removing the instrument. When the orientation is learned in the dorsal and dextralateral positions the use of this technic has proved to be of considerably greater value than the use of the left side position alone in general use. The fluoroscopist

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does not consider that his examination is adequate unless he has rotated his patient through both obliques, the posterior and the head-low positions. The gastroscopic blind areas are often improved and sometimes eliminated entirely with this technic. In many instances we have seen lesions using this method which could not be seen with the single position technic. In other instances the additive value of seeing a lesion from different angles has made it possible to make a definite diagnosis rather than a questionable one. This may be illustrated by the ability to see depth in an ulcer which might otherwise appear to be a "fleck of mucus" or it may be possible to distinguish between a fleck of mucus and a superficial gray erosion. The method has been confined to the use of instruments with flexible portions of standard length and is not recommended for use with rigid instruments or those with long rigid portions. I will illustrate with a short movie some of the technics and types of instruments recently developed to improve the mechanical or technical problem of "blind spots" in gastroscopy. A few slides will illustrate briefly the advantages of multiple positions in gastroscopy. Displacement of the gastric wall due to gravity effects undoubtedly account for improvement in view when the patient is shifted to the dorsal and other positions. In the case of W. S. only a suggestion of an ulcer at the angulus was seen in the standard left side position, but it became progressively more visible as the patient was rotated to the dorsal and then to the dextralateral position. The ulcer which had been diagnosed benign by x-ray and barely seen in the standard position gastroscopically was seen to be a malignant ulcer by the "rotation" or "multiple positions" technic. In the case of P. S. an ulcer as well as the contracting pylorus could be seen in the same view with the patient in the dorsal position. In the standard left side position this view was not possible. Here the composite picture made possible by viewing the ulcer at different angles clinched the diagnosis of benign ulcer. In the case of C. H. two bleeding gastrojejunal ulcers were seen on six different occasions in the dextralateral position but were never seen in the left side position. This was an important bleeding case following gastric resection in which gastrojejunal ulcer or postoperative gastritis as the cause of the hemorrhage had to be differentiated. In the case of L. R. "ball-valve" obstruction at a gastrojejunal stoma by a benign tumor was found. Here the additive value or effect of rotation made it possible to see that this small tumor impinged on the edge of the stoma so that it might occasionally block the opening and account for the intermittent vomiting which was patient's principal complaint. Having been found to be benign by frozen section the tumor was removed by simple excision rather than extensive gastric resection. This operation was followed by complete recovery. In some instances a posterior wall polyp or gastritic lesion or gastrojejunal stoma is seen only by the use of multiple positions as shown in the cases of R. P., A. C. and L. H.

All that has been illustrated up to this point has been primarily concerned with recent developments in solving or improving the technical imperfections of gastroscopy. Namely, trying to make it possible to always see the lesion. Other

simple technical impediments such as improper emptying of the stomach and inadequate psychological preparation of the patient obviously may also account for a lesion not being seen. Manual pressure upon the external abdominal wall with the instrument *in situ* may occasionally improve a blind spot.

Newer Developments in Diagnosis:—Now let us consider the second phase of the trends toward improvement in gastroscopic diagnosis. Namely, trying to make it possible to always diagnose the lesion when seen. Observations in our clinic and in some of the other larger centers indicate that nearly two-thirds of the incorrect diagnoses in gastroscopy when the lesion is seen are about equally divided between: 1. Failure to correctly differentiate between benign and malignant ulcer and, 2. Failure to correctly differentiate between hypertrophic gastritis and malignancy. What is being done for the solution of this important problem? Perhaps the most important single development in this direction is the availability of gastro-biopsy. Just as punch biopsy of the liver has improved our concepts of hepatitis and other liver disorders so improvements in diagnostic concepts are developing as the result of gastro-biopsy. There are two types of instruments now in use. The one which is in production is the Benedict instrument made by the American Cystoscope makers. It is built on the principle of the "alligator-jaw" mechanism. Unquestionably it carries some risks over and above that of the simpler instruments, and unfortunately all portions of the stomach seen cannot be reached with the biting forceps. As a word of caution, I think that only gastroscopists who have had considerable experience with the simpler gastroscope should attempt to use the biopsy instrument. The other instrument described by Dr. John Tomenius of Stockholm and others makes use of suction to draw portions of the mucosa through holes or apertures in a rubberized tube. The tiny sucked-in portion is snipped off by a sliding cylinder with a sharp-edged circumference which operates inside the tube. This may have advantage as a safety factor. However, it has the disadvantage of not permitting the operator to see the lesion at the moment of making the biopsy. When using this instrument, gastroscopy is carried out first. The area to be biopsied is located and studied in the gross. The gastroscope is then removed and the biopsy tube inserted so that the aperature corresponds to the position previously occupied by the gastroscopic eye or objective. The latter method is being used in Sweden and with modification in England also. The illustrations shown here of the assembled instrument, the disassembled instrument, and 4 photomicrographs of biopsies were sent to me by Dr. Tomenius from Stockholm, Sweden, to be shown at this time. I think that the photomicrographs indicate that excellent and adequate biopsies can be obtained with this instrument. Obviously the last word has not been written or spoken with reference to the histopathology of tissue removed by gastro-biopsy. Perhaps these newer trends and technics may represent an important approach to the solution of such problems as the earlier diagnosis of gastric cancer; the relationship of chronic gastritis and cancer; the relationship of ulcer and cancer, etc. Certainly gastro-biopsy and the improvement of view of gastroscopic "blind spots" should lead to the establishment of new diagnostic criteria for x-ray and gross gastro-

scopic diagnosis. This may turn out to be perhaps the most important contribution of gastro-biopsy. The next case will illustrate the importance and the difficulty of these problems. A female patient age 45 yrs. presented a typical clinical story of peptic ulcer. The x-ray diagnosis was prepyloric ulcer probably benign. Antral deformity was shown but a typical ulcer niche could not be shown on the film. The patient was therefore gastroscoped and extensive hypertrophic gastritis was seen. The ulcer crater could not be seen. Gastric acidity was high. Rigid medical management was instituted and the symptoms were greatly improved after 2 or 3 weeks but tended to recur when the medication was interrupted. Reexamination by x-ray after about 5 weeks still showed a bizarre deformity of the antrum, again interpreted as an ulcer. Because of the patients age and a slow heading gastric lesion surgery was performed. The gross specimen again showed extensive hypertrophic gastritis and in addition partially healed prepyloric ulcer. Usually a gastric ulcer associated with extensive hypertrophic gastritis is regarded as being most likely benign. The microscopic section in this case revealed infiltrating carcinoma and a carcinomatous ulcer. The section shows carcinoma cells infiltrating along the submucosa immediately beneath typically appearing hypertrophic gastritic nodules.

This case serves to indicate not only the need for further development of the fertile field gastro-biopsy, but the need for more comparative studies of roentgenographic, gastroscopic and pathologic patterns of gastric disease. Any discussion of advances in gastroscopy should allude to the findings of comparative studies of this type. A few reports of fairly large series of cases comparing roentgenographic and gastroscopic diagnoses have appeared in the literature. In these reports the final diagnoses have been confirmed by surgical or postmortem examinations. It has been found repeatedly that while many cases which are diagnosed by gastroscopy are more conveniently diagnosed by x-ray, 15 to 25 per cent of cases missed by x-ray may be diagnosed by gastroscopy. For example: In a series of one hundred proven cases of gastric cancer studied at the Mayo Clinic by Moersch and Kirklin[2] diagnostic accuracy was improved by 22 per cent by the added use of gastroscopy. That is to say that x-ray was wrong in diagnosing carcinoma of the stomach in 42 per cent of 100 cases proven by surgery and postmortem examinations. Gastroscopy diagnosed 20 of the cases missed by x-ray so that with the two methods diagnostic accuracy was increased from 42 per cent to 62 per cent.

In a series of 125 proven cases of gastric carcinoma all of whom had x-ray and gastroscopy studied by Benedict[3] at the Massachusetts General Hospital, diagnostic accuracy was improved 16 per cent. That is to say 20 out of 125 cases were diagnosed only by gastroscopy.

In our own series being analyzed from a group of 2,500 consecutive gastroscopies a preliminary analysis reveals figures within the same percentage range as previously reported. It is therefore obvious that in spite of the inconvenience and imperfections of gastroscopy, the diagnosis of gastric cancer has been notably improved by its widespread use. I would say that present trends indicate that gastros-

copy has a great future as an important adjunct in the diagnostic armamentarium of the gastroenterologist and that it will contribute significantly to the better understanding of the physiology of gastric disease.

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SOURCES OF ERROR IN THE RADIOLOGICAL DIAGNOSIS OF GASTROINTESTINAL TRACT DISEASE *

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Radiology is indispensable to present day gastroenterology. X-ray study is, perhaps, the most important single examination in the diagnosis of gastrointestinal tract lesions, and is admittedly a part of the work-up of almost every patient suspected of harboring disease of the alimentary tract.

While admitting the importance of radiological evaluation, we must, at the same time, acknowledge its limitation and errors. An effort to evaluate the causes or sources of radiological errors may lead to attempts to correct these errors or reduce their incidence.

Experienced radiologists and clinicians are often in considerable disagreement as to the reliabilities and accuracies of roentgen diagnosis. Sosman[1] has recently attempted to evaluate the specificity and reliability of diagnosis made primary by roentgen procedure. In the gastrointestinal tract he considered that competent radiological study could demonstrate 80 to 85 per cent of significant lesions and to specify their identity in 90 per cent. It is my firm belief that such percentages of accuracy are obtained only by well trained and competent radiologists.

The significance of certain roentgen observations may be subject to varying interpretations. Such findings as tertiary esophageal contractions, temporary pylorospasm, minor delays in gastric emptying, slight to moderate variations in the small intestinal pattern and motility, prolonged retention of barium in the appendix, and long delay in gallbladder emptying are illustrations where radiologists may either stress or discount the importance of such findings.

Indeed, there are anatomic situations where the radiologist may consider a finding as essential normal, or a normal variation, or he may consider it of clinical importance depending upon his concepts and teachings. Such situations include small gastric hiatal hernias in the elderly, especially when producible only with markedly increased intraabdominal pressure. So-called gastroptosis and variations in the position and appearances of the duodenal loop with slight degrees of proximal loop dilatation, may fall into this category. The large dilated gallbladder with slow rates of emptying may be subject to various interpretations by various observers. It is recognized, of course, that the final evaluation of the significance of some of these observations must be made by the correlation of all clinical and all radiological findings by a trained and competent clinician. Nevertheless there is considerable opportunity for the radiologist to ignore or emphasize many observations of questionable significance, and in so doing to possibly err either in a positive or negative sense in his analysis of the presence or absence of pathological physiology or of actual pathology.

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All of the above brings us to the philosophy of roentgen interpretation and diagnosis. Rigler[2] has emphasized the factors involved in the limitations of radiological diagnosis of chest lesions, namely: the time factor or the age of the lesion, the size of the lesion, and the time relation of visibility of the lesion to symptoms or signs. To these three factors of time, size, and time relationship, Sosman[1] has added a fourth, namely: the frequency of any given lesion for the age, sex and race of the patient, and the location, distribution and x-ray appearance of the lesion. Such a fourth factor emphasizes mathematical probabilities in a given situation.

Definite diagnosis of the specific nature of a gastrointestinal tract lesion is not always possible. We are fully conscious of the fact that surgeons and pathologists have frequent difficulty offering anything more than a suggestion as to the pathological nature of a lesion under direct palpation and vision. Pathologists may be undecided on histological section as to the inflammatory or neoplastic nature of certain lesions or as to their benignity or malignancy.

Errors of radiological interpretation may occur when too persistent an attempt is made to specify the exact nature of gastrointestinal tract lesions. While there are frequent instances where the radiologist can exactly identify the nature of the lesion, there are numerous occasions when differentiation between several or more possibilities is impossible. Similarly there are occasions where one can in no way be *sure* of the existence of a pathological process. In such instances a report of "possible" or "probable" lesion is desirable.

A reasonable philosophy of roentgen interpretation is essential for the radiologist and clinician alike. Such a philosophy must include all degrees of surety of diagnosis from "unquestionable" through "highly probable", "probable", "indefinite suggestion of", and "negative". Qualifying notations ranging from "undoubtedly significant", "probably significant", "possibly significant", "probably of no clinical significance" and "not clinically significant" are valuable additions to comments regarding certain variations from norm, and should be part of the philosophy of radiological evaluation and interpretation of case problems. Adequate roentgen interpretation of the polymorphous appearance of the gastrointestinal tract requires a more or less detailed knowledge of all clinical data. Attempts at interpretation without this data are, in the author's opinion, usually unsatisfactory.

Sources of Error In Roentgen Diagnosis

While errors in diagnosis may be either positive or negative, the causes of both are usually the same.

Admitting that there are certain instances in which a gastrointestinal tract lesion exists, but it is of too small a size for roentgen demonstration, the majority of gastrointestinal tract organic lesions are capable of roentgen detection. Error or failure to demonstrate a lesion, amenable to demonstration, is usually due to one or more of the following factors:

1. Inadequate clinical data.
2. Incorrect decision as to the type of examination.

3. Inadequate preparation of the patient.
4. Inadequate preparation of the examiner.
5. Inadequate equipment.
6. Inadequate examination.
7. Incorrect interpretation of findings.

We note that of the seven listed factors, five are inadequacies. It is my opinion that improperly performed examinations account for the majority of errors in roentgen diagnosis. Not seeing by not properly examining is more important as a source of error than incorrect interpretation. A proper roentgen examination presumes a radiologist to be equipped with the "know how" and with the knowledge of how, when and where to examine.

Inadequate Clinical Data

The competent gastrointestinal radiologist should, first of all, be a good clinician with a fair knowledge of the normal and variations of the normal, of the gastrointestinal tract. Knowledge of the incidence, probability and time relationship of gastrointestinal tract lesions to symptoms is a prerequisite of his training. In short, the clinical radiologist should be somewhat of a gastroenterologist. Lack of essential clinical data handicaps the performance of a proper gastrointestinal study. Altered appearance as the result of previous known disease, or of operative intervention, can only be correctly interpreted with full knowledge of the patient's previous history. Where to search most diligently in the twenty or more feet of gastrointestinal tract is judged on the basis of complete clinical data.

Incorrect Decision As To The Type of Examination

It follows that incomplete clincial data may lead to an incorrect decision as to the type of examination to be undertaken. Not infrequently small intestinal lesions, particularly in the lower small intestine, can be discovered only by either a detailed progress meal study of the small intestine, or a so-called small intestinal enema. Since the routine so-called G.I. series does not usually include such detailed small intestinal study, a considerable extent of the gastrointestinal tract may be inadequately studied by a routine G.I. series. The suspicion of colonic polyposis usually indicates the desirability of so-called double contrast enema study in addition to routine barium enema study. The type of examination to be employed is usually of great importance.

Inadequate Preparation of The Patient

Even the ingestion of a small amount of liquid prior to roentgen study of the stomach, may seriously interfere with satisfactory mucosal pattern visualization. The practice of administration of gallbladder emptying stimulants for cholecystographic study only a short time before upper gastrointestinal tract study is probably inadvisable. Satisfactory mucosal pattern visualization may not be obtained. The examination of a patient whose stomach carries a large amount of gastric secretion may be quite inadequate. It is probably advisable to empty the stomach

by intubation in these cases. Poor or inadequate preparation of the colon by ineffectual attempts at catharsis is a not infrequent cause of missed colon lesions.

Inadequate Preparation of The Examiner

Inadequate dark adaptation preliminary to attempting fluoroscopic procedures is inexcusable. I have had the personal experience of missing lesions in the first of several series of fluoroscopic observations simply because of inadequate dark adaptation. The curves of dark adaptation are well known. A minimum of at least twenty minutes of red goggle eye coverage is a necessity.

Inadequate Equipment

Gastrointestinal study attempted with antiquated equipment is still a common source of error. A tilt-top radiographic fluoroscopic table capable of tilt from upright to at least some degree of Trendelenberg position is a necessity. Compression spot filming devices are common to all present-day well made equipment. Without spot filming certain lesions either can be missed or inadequately demonstrated. Serialographic radiography is a necessity in many cases. Equipment must be of sufficient power for adequate radiography of the heaviest of patients.

Inadequate Examination

A hastily performed study is often worse than no study at all. The demonstration of esophageal varices often times requires a meticulous and most careful examination. A combination of careful fluoroscopy, spot films and serialographic studies in erect and recumbent positions, and including various oblique, lateral and Trendelenberg positions, constitute a thorough study of the upper gastrointestinal tract. Procedures with reference to the small intestine must, at times, include progress meal studies both by film and fluoroscopy, small intestinal enema studies, and, on occasion, small intestinal intubation studies. Occasionally, the only satisfactory visualization of the distal ileum is obtained by barium enema ileocecal reflex of barium. Colonic examinations may, or may not, require a detailed procedure. Repetition of barium enema and double contrast enema on several occasions is at times necessary before smaller polyps of the colon are demonstrated.

Incorrect Interpretation of Findings

Following demonstration and visualization of a gastrointestinal tract abnormality or lesion, its correct interpretation depends upon the training, experience, skill and ability of the radiologist. While the criteria for the differentiation of benign from malignant ulcerative lesions of the stomach is not absolute, certain signs or sign complexes offer valuable leads as to this differentiation. A knowledge of the numerous variations in the appearance of mucosal folds is important in differentiating mucosal lesions from normal mucosal variations. The so-called "stellate impression" of the esophageal gastric junction and a somewhat similar impression of the ileocecal valve area may lead the inexperienced observer to an incorrect interpretation of a pathological process. Interpretation is largely a function of the observer's training and experience.

Summary

Radiological diagnosis in competent and experienced hands has a reasonably high index of accuracy. An attempt has been made to analyze the possible sources of error in roentgen diagnosis.

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Discussion

Dr. I. Snapper:—In this part of the world—it doesn't hold true for the whole world—tuberculosis of the intestine should be diagnosed only if there is at the same time radiologically active tuberculosis of the lungs. This is important because other granulomas of the intestine like Boeck's sarcoid, or regional ileitis, or schistosomiasis present on the x-ray the same picture as tuberculosis. In case a patient has no radiologically active tuberculosis of the lungs, then one of the nontuberculous granulomas just mentioned, are more probable.

Sprue is a disease which occurs in the tropics, characterized by fatty diarrhea, abdominal distention and macrocytic anemia. These patients react readily on certain diets and liver treatment.

In this part of the world fatty diarrheas of unknown etiology are also frequently encountered. This disease should be carefully distinguished from tropical sprue, because fatty diarrhea which occurs here hardly ever reacts on the treatment which is so beneficial in tropical sprue. In addition patients with nontropical steatorrhea of unknown origin have usually clubbed fingers, patients with tropical sprue never.

Whether the designation nontropical sprue is a correct one, seems at least doubtful.

The differential diagnosis between hypertrophic gastritis and carcinoma of the antrum is a very difficult one. I know that in many cases experienced radiologists have hit on the right diagnosis and have correctly diagnosed "antritis", or "hypertrophic antrum gastritis", but nevertheless when the slightest doubt exists surgical exploration is indicated.

I fully agree that x-rays of the surface of the mucous membrane of the colon are more important for a correct diagnosis than x-rays of the colon, overfilled with barium. The carcinomas of the lowest part of the sigmoid just beyond the reach of the sigmoidoscope are the most difficult to diagnose. For this part of the colon not only A.V. photographs, but special lateral positioning is necessary.

TREATMENT OF AMEBIASIS*

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Amebiasis is caused by infestation with ameba histolytica. The motile forms of the amebae, the so-called trophozoites, are tissue parasites and live in the submucus layer of the intestine and in adjoining structures, especially in the liver. As soon as the environment becomes unfavorable to the amebae, cyst formation takes place. In order to cure amebiasis the treatment must be directed not only against the trophozoites but also against the cysts. Many discrepancies in the results of treatment can be explained because this fact has not always been sufficiently appreciated.

Amebae cause tissue necrosis, no actual suppuration takes place. Thus amebiasis of the rectum if viewed through the sigmoidoscope presents itself as small elevations with necrotic centers and not as large ulcers as seen in ulcerative colitis. It also follows that if amebae penetrate into the liver no actual abscess is formed but necrosis results. The so-called tropical liver abscess thus consists of a large cavity filled with necrotic liver tissue. Although amebiasis ultimately involves the whole colon, the disease actually starts in the cecum. The amebae are transported from the cecum via the branches of the portal vein to the liver. Since blood from the superior mesenteric vein which drains the ascending colon is directed mainly towards the right lobe of the liver, for the most part, amebic liver abscesses are located in the right lobe of the liver.

It must be emphasized that the treatment of amebiasis in the United States offers less difficulties than when amebiasis must be treated in tropical areas. The better nutrition of the Westerners probably plays an important role in the explanation of this difference. It is a general rule that the treatment of parasitic infestations is unsuccessful as long as the patient is under-nourished, but that worm infestations are hardly ever clinically important when the general nutritional condition of the patient is improved.

Until 1912, the treatment of amebiasis was very unsatisfactory indeed. In the beginning of the 17th century the Hollanders captured Brazil which at that time was in Portuguese hands. A Dutch army physician, Willem Piso, described in his report on his experiences in Brazil a remarkable root which the natives used successfully in the treatment of bloody diarrheas. This "radix mirabilis" was the radix ipecacuanha which in the 18th, 19th Centuries and in the first decade of the 20th Century was mainly used as an expectorant, occasionally as an emetic. In 1817 Pelletier isolated a glucoside from this radix ipecacuanha, which if given by mouth worked as an emetic and was therefore designated as emetin. Only 100 years after the isolation of this emetin, Vedder discovered that this substance killed ameba histolytica in the test tube. Rodgers in 1912 injected emetin into patients with amebiasis and found that after 10 daily injections of 1 gr. of emetin hydrochloride

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the signs of amebiasis disappeared. Younger physicians can hardly understand the enthusiasm which this discovery evoked in areas where dysentery was endemic. Patients who for years had been suffering from this debilitating disease and who were evidently lying at death's door were resuscitated by this simple treatment. The same holds true for patients suffering from acute amebiasis with large numbers of hemorrhagic stools and high fever. The glowing enthusiasm for the treatment of amebiasis by emetin decreased somewhat when further experiences showed that these cures were usually only temporary; after one or more months the disease often recurred. In the hope that larger doses of emetin would actually prevent the development of these recurrences much more than 1 grain daily for 10 days were administered. Then it appeared that in large doses emetin is a dangerous drug and can give rise to cardiac death. These unfortunate accidents have not been forgotten and the fear for emetin has persisted. However, clinical experience has shown, and careful investigation has confirmed, that ten injections of 60 mg. of emetin hydrochloride are without danger and do not give rise to any cardiac complications. Nevertheless it is wise to measure the blood pressure of patients receiving emetin injections and to stop the treatment, at least temporarily, when the blood pressure falls during therapy. The most important danger signal consists of a heavy sensation of fatigue in the shoulders, arms, and sometimes in the legs. When this sign develops, emetin must be stopped even if the blood pressure has not fallen.

It soon became evident why emetin has only a temporary effect. Emetin kills the trophozoites but does not touch the cysts. Therefore, the acute signs of amebiasis which are due to the action of trophozoites disappear, but after some time new amebae develop from the cysts and a recurrence of the disease sets in. It was therefore necessary to employ a drug which not only kills the trophozoites but also kills the cysts. Very shortly after the introduction of emetin as a remedy against amebiasis it was shown that certain organic compounds which contain iodine can be used for this purpose. Yatren, nowadays designated as chiniophone, and vioform destroy amebic cysts. In later years a new iodine compound, diodoquin has been introduced. One chiniophone tablet of 250 mgs. is usually given 4 to 6 times daily for 14 days or two tablets of diodoquin 3 times daily for 3 weeks. Diodoquin is tolerated extremely well and is less prone to give rise to diarrhea, a complaint frequently encountered in patients treated with chiniophone. In general it may be said that nearly all cases of amebiasis regardless of whether they are acute or chronic, whether trophozoites or cysts are prevalent, are cured by a diodoquin series as mentioned above.

This treatment is so simple and so devoid of undesirable side actions that for years we have not employed carbarsone at all. This pentavalent arsenical preparation given as tablets of 250 mg. twice daily for 10 days clears up many cases of amebiasis. However it seems wise to avoid the administration of arsenic unless it is absoluately necessary.

The iodine compounds as chiniophone, vioform, and diodoquin are not absorbed from the intestinal tract and therefore do not influence amebic lesions located out-

side of the intestine. Consequently, they have no curative action upon amebic hepatitis or amebic liver abscess. Therefore, emetin injections remain the treatment of choice, in amebic hepatitis or tropical liver abscess, because diodoquin treatment alone would leave the hepatitis untouched. However, in this climate amebic hepatitis and liver abscess are relatively rare and thus in the Western part of the world in patients with amebiasis without clinical signs of liver involvement diodoquin treatment is sufficient almost without exception.

The surgical treatment of amebic liver abscess was, at least before the antibiotic era, frought with a heavy fatality of about 30 per cent. This is the reason why in the Orient physicians favored puncture of the abscess with paracentesis and emetin therapy as the treatment of liver abscess. It is usually necessary to repeat the paracentesis twice or three times at three-day intervals. This paracentesis treatment combined with emetin has hardly any fatality and leads almost without exception to a cure of the abscess. Only once have we been obliged to invoke surgical help. In this case the liver necrosis had not liquefied but had remained as a large mass of necrotic tissue within the abscess cavity. This large mass could not be evacuated through a paracentesis needle and surgical drainage was necessary in order to obtain a cure.

Textbooks mention that the pus of a liver abscess is chocolate in color. This is only partially true. Upon puncture of an amebic liver abscess a red fluid is obtained. Since the liver parenchyma itself has such strongly reducing properties the red puncture fluid soon becomes chocolate brown due to the formation of methemoglobin. The blood-like material which enters the syringe can immediately be distinguished from true blood by evacuating the syringe into a small basin. Liver blood coagulates immediately whereas the pus of an amebic liver abscess remains fluid. In this way the inexperienced operator can recognize the material obtained as liver "pus" and allay his fears that he has punctured a large branch of the portal vein. Amebae live in the abscess membrane. Therefore, when a liver abscess is punctured amebae are often not present in the central semi-liquid substance which is removed. After the abscess has been evacuated the next day another puncture should be performed and in this specimen amebae are usually found. Due to the changes in pressure within the abscess cavity, the amebae leave the abscess membrane and enter the abscess cavity.

Occasionally, cases of amebiasis have been encountered with fistulae remaining after operation for liver abscess which have resisted the usual emetin, iodine, or arsenic treatment. Fortunately, several of the modern medicaments which have been developed in later years are also amebicidic in nature. Good results have been reported of chloraquin treatment of such resistant cases of amebiasis. Four tablets containing 250 mg. of chloraquin diphosphate are given on the first day, and two such tablets are administered on the 2nd, 3rd, and 4th day.

The therapeutic action of chloraquin in amebiasis may shed a new light on the favorable results obtained with chiniophone, vioform, and diodoquin. It was generally accepted that the latter three drugs were active because they contained considerable amounts of iodine. However, these three iodine compounds are at

the same time quinoline derivatives. In view of the action of diodoquin in amebiasis, the question seems justified whether a good deal of the therapeutic activity of the three iodine compounds may not depend upon the quinoline ring of the molecule.

Three modern antibiotics can also be used for this purpose. Both aureomycin and terramycin capsules in doses of 250 mg. four times daily administered for one week and bacitracin 30,000 units every six hours for five to twenty days have received favorable comments in the treatment of amebiasis. Whereas emetin is directly amebicidal, the action of aureomycin and bacitracin is mainly indirect. Both of these antibiotics change the flora of the intestine by killing off most of the clostridia and enterococci. It seems that the main part of the food upon which amebae live consists of these bacteria. In this way it becomes evident that sterilization of the intestine by these modern antibiotics can have a favorable influence upon amebic infestation. The possibility that these antibiotics have at the same time a weak amebicidal action has also to be considered.

Summary

1. For the treatment of amebiasis without amebic hepatitis or amebic liver abscess peroral treatment with an organic iodine compound (diodoquin, chiniophone, vioform) seems to be the treatment of choice.

2. As soon as amebiasis is complicated by hepatitis even without abscess formation, emetin treatment must be added to the above.

3. Treatment of amebic liver abscess should consist of repeated paracentesis of the abscess cavity and parenteral emetin.

4. In cases of amebiasis, resistant to organic iodine compounds and emetin, chloraquin, aureomycin, terramycin, and bacitracin can be used.

Question:—Did you always find an organism in these live abscesses? Some of them are sterile, I understand.

Dr. Snapper:—Amebae are tissue parasites: they live in the tissues. Therefore, the amebae are mainly present in the abscess membrane. If the abscess is punctured for the first time fluid from the central part of the abscess cavity is obtained which often does not contain amebae. When the puncture is repeated on the next day, the amebae have left the abscess membrane due to the vacuum caused by the first paracentesis, and on the second day the amebae are nearly always present in the punctured material.

Formerly in 50 per cent of amebic liver abscesses, no amebae could be found in the stool. Nowadays, thanks to the cultures of amebae from the stools this percentage must be much lower.

Question:—In the case of a person who contracts amebiasis, can he rid himself of amebae if he recovers untreated?

Dr. Snapper:—He can get rid of the trophozoites but not of the cysts, and thus danger for recurrence always remains. Such a cyst carrier is also dangerous for his contacts because the infection is only transmitted from carrier to contacts by transmission of cysts.

Question:—You said you would mention a word about culture of the cysts.

Dr. Snapper:—Different media are available for the culture of amebae and cysts. Dr. Spingarn, in our laboratory, usually employs a liver infusion agar slant over which a mixture of horse serum and normal saline is spread. The amebae feed on bacteria, but on the other hand, too many bacteria overgrow the amebae and kill them. Antibiotics must, therefore, be added to the medium, but only in small quantities, because the anaerobic bacteria, like clostridium should not be killed. The best results are obtained by the addition of small amounts of penicillin, or better even, of streptomycin. Finally, rice powder must be present in the cultures because the latter increases the growth of the amebae.

Question:—What degree of specificity has the complement fixation test for amebiasis?

Dr. Snapper:—In my experience this test is not very specific; and, secondly, this test has lost part of its practical significance because in actual amebiasis the amebae can nearly always be found by culture.

Question:—Would you say something about amebic arthritis?

Dr. Snapper:—Arthritis due to dysentery bacilli is frequent, but amebic arthritis is rare.

Question:—Do you have to have an intestinal condition to have a liver abscess?

Dr. Snapper:—There must have been an intestinal amebiasis originally, but the intestinal infestation may have cleared up either spontaneously or because the patient received a certain amount of amebicidic treatment.

Appendicitis due to amebiasis is rather dangerous, because these patients often develop pylephlebitis.

HEMORRHOIDS

Etiology, Pathology and Treatment

(Part I)

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Hemorrhoidal disease, antedating the history of medicine, is the commonest of rectal disorders and over the centuries it has been subjected to much exploitation and misdirected therapy. Under the facile diagnosis of "piles" there has masqueraded both for the practitioner and patient a variety of benign and malignant anorectal as well as gastrointestinal, gynecologic and urological conditions. It is one of the commonest self-diagnosed, self-treated and universally maltreated of all human diseases.

The bulk of the medical literature on hemorrhoids has been concerned with the treatment which has been controversial and carried on with inadequate evaluation of the extent of the disease, its etiology and its commonly associated pathology. Moreover, the large variety of surgical procedures have been and still are performed with but little regard for the anorectal anatomy as well as the physiology of defecation.

Few operations in the history of surgery have been performed more often, for symptoms rather than actual pathology, with more variations; new methods, under more adverse conditions; with more questionable indications; more postoperative pain, than has hemorrhoidectomy, and with all a low mortality. This is an incredible tribute to the human anal canal.

The injection, nonoperative or ambulant treatment of hemorrhoids has been a highly provocative subject. Since its initial use in this country, presumably by Blackwood in 1866, the method was widely exploited and commercialized by the so-called "pile doctors". At the same time it was generally discredited by the general practitioners and particularly the general surgeons.

Condemnation of the injection method by the medical schools and practitioners was not based on scientifically controlled observation and to inject hemorrhoids was quackery without further ado. And so the "pile doctors" thrived on the profession's aloofness. The unfortunate and discrediting aspect of this controversy was the large number of hemorrhoidal patient's sacrificed with premalignant and malignant conditions without a comprehensive proctologic or other indicated examination. This condition still persists.

Largely to the credit and the influence of the English Proctologists and the American Proctologic Society, the injection treatment of internal hemorrhoids has been placed on a sounder and less controversial basis. The business of medicine will probably never be entirely divorced from the art of medicine, but with an increasing interest in proctology, with better teaching methods, improved technics and at long last the recognition of a separate Certifying Board in Proctology the therapy of hemorrhoids will become more scientific and "pro bonum publicum".

It is hoped that this presentation may contribute constructively toward this objective.

Definition

The term hemorrhoid is derived from the commonest symptom of hemorrhoidal disease. As generally used in medicine it refers to varying degrees of tumefaction in either of the hemorrhoidal plexuses and their communicating radicals, usually those internally. Perianal hematomata, skin hypertrophies, enlarged papillae, are loosely referred to as hemorrhoids.

The laity commonly refers to any hemorrhagic or protruding anorectal condition as hemorrhoids or "bleeding piles". The various symptoms arising in the clinical course of hemorrhoids is referred to as hemorrhoidal disease.

Incidence

Although observed at all ages, hemorrhoids are essentially a disease of adult life, predominating in the third and fourth decades, the active sexual, physical and mental period of adjustment.

In infancy, Shapiro, in 547 proctologic examinations, observed hemorrhoids in 18 instances, 8 internal, 7 external and 2 of the combined variety. In the younger age groups the hemorrhoids are usually of the soft vascular nonprolapsing type while as age progresses the prolapsing and mixed variety are more commonly observed.

Statistics, without reference to the variety, indicates that they are somewhat commoner in the male sex, a ratio of about 5 to 4.

Etiology

An incredible variety of predisposing as well as exciting causes have been advanced for hemorrhoids. Only the more important will be discussed.

Heredity, as frequently emphasized, is undoubtedly of etiological significance particularly in early adult life. Similar environmental, dietary, occupational and neurocirculatory factors are perhaps of some significance but a congenital deficiency in the supporting tissues of the hemorrhoidal areas is perhaps of greater importance.

Anatomical factors are unquestionably of significance. These include the upright position of man, the portal venous drainage, the absence of valves in the hemorrhoidal veins and finally, the looseness and mobility of the hemorrhoidal plexuses which provide a physiologic "venostatic" mechanism for the anal canal.

Exciting causes which are commonly mentioned and repeated in the literature are improper diet, drugs, occupation which require straining with increased abdominal pressure, etc. The most important and prevalent causes are chronic infection and irritation to which the internal hemorrhoidal plexus in particular, is more or less constantly exposed.

The repeated vasocellular reaction to infection and congestion, active or passive, results in lymph stasis, cellular sludging, histaminia, and histanoxia in the

hemorrhoidal plexuses. These pathogenic factors produce a fragmentation and disintegration of the supporting matrix and a dilatation and fragility of the veins. Infection and histaminia are the more important factors which are activated and accentuated by conditions which produce repeated vascular congestion in the hemorrhoidal plexuses. Such conditions, may be local or remote and they are commonly referred to in the etiology as mechanical or physiological causes.

The mechanical causes may be intrinsic or extrinsic. The former include constipation, obstipation, prolapse, tumors, etc. The latter, pregnancy, uterine and adnexal tumors, prostatism, etc. Of the physiological causes those producing excessive and repeated increase in abdominal pressure, portal congestion, hepatitis, cardiac failure, etc., are the commonest.

It may be stressed that infection of the overlying hemorrhoidal mucosa with erosion and superficial ulceration is important etiologically as well as symptomatically because it accounts for the bleeding, the impellent sign. Infection directly into the hemorrhoidal stroma is secondary. The crypts have been stressed as an important channel in hemorrhoidal infection, however, it should be noted that the crypts drain into the pecten rather than into the submucus space which contains the internal hemorrhoidal plexus. The liberation of histamine and histamine-like bodies is probably of greater significance in the etiology of both internal and external thrombotic hemorrhoids than heretofore recognized.

Classification

Hemorrhoids may be classified anatomically according to the venous plexus involved into:

1. External
2. Internal
3. Mixed

1. *External Hemorrhoids* may be further classified into varicose (rare), thrombotic (common), (Perianal hematomata) and skin tags; erroneously referred to as hemorrhoids.

2. *Internal Hemorrhoids* may also be subdivided into prolapsing, thrombotic and strangulated.

3. *Mixed Hemorrhoids* include a combination of internal and external varicosities with enlarged interconnecting veins and hypertrophy of the supporting stroma, usually with a skin tag. Qualifying or descriptive terms have been applied to this classification based primarily on prominent pathological changes or complications which may be of significance in the therapy. These include, capillary, nevoid, arterial, fibrous, thrombosed, white, columnar, inflamed, "squamitized", constitutional, etc., hemorrhoids.

Symptoms and Diagnosis

General considerations:—The course of hemorrhoidal disease varies in different individuals. In some cases long periods of latency vary with occasional acute symptoms of minor complications, external thrombosis, partial prolapse,

pain, edema, pruritus and particularly bleeding, etc. Bleeding usually provokes medical consultation for fear of malignancy.

In others the hemorrhoidal pathology is well established but bearable to the patient by self-palliation, manual reduction, habitual catharsis, use of ointments, suppositories, etc., etc. Hemorrhoidal disease is usually only recessive when the causative factors are of short duration as in pregnancy, fecal impaction, uterine or prostatic tumors, etc.

When not under pressure hemorrhoids usually appear as slightly enlarged folds of the perianal skin. Under obstructive pressure, straining, defecation, etc., however, they may assume a ballooned-out appearance and form a coronal, bluish tinged, bulbous swelling surrounding the anus. They are common in geriatric practice. They may form the external vascular element of the intero-external or mixed variety of hemorrhoids. During operative procedures on the internal hemorrhoids their full extent usually becomes evident.

True varicose external hemorrhoids are usually asymptomatic or only produce a feeling of fullness during defecation. They are diagnosed by direct inspection and require sufficient increase in venous pressure to fully reveal their extent. Treatment is along prophylactic and palliative lines with special attention to conditions producing increased portal or abdominal pressure and correction of obstructive and prolonged defecation.

If radical measures are considered advisable because of repeated thrombosis or a persistent complaint of fullness on standing, straining, coughing and following defecation, the simplest procedure is to puncture the veins with the actual cautery or to unroof them by partial excision. When they form the external component of the mixed hemorrhoid they should be excised with the internal hemorrhoid. These varices should under no consideration be injected with a sclerosant.

External Thrombotic Hemorrhoids (*Perianal Hematomata*):—Erroneously referred to as an external thrombotic hemorrhoid, perianal hematomata result from a rupture of one or more of the external hemorrhoidal radicals. They are the commonest variety of the external group. They vary widely in size, from minute millet seed clots which are commonly undiagnosed and are more or less asymptomatic, to large hematomata. These may be multiple. The size probably depends on the number of veins ruptured and the duration and force of straining which usually precipitates venous rupture or extravasation. The extravasated blood rapidly becomes encysted in the perianal tissues and surrounded by an encysting membrane, not the vein wall (Fig. 1). No satisfactory reason has yet been advanced for single or multiple rupture of these veins but it is not unlikely that the action of histamine or a histamine-like body is the underlying factor predisposing to an increased capillarity or permeability. It is difficult to explain the simultaneous rupture of a large number of these venules, sometimes producing a complete painful shotty, edematous perianal swelling.

The single hematoma are usually lateral. Those occurring in the commissure are apt to be more painful and provoke considerable sphincter spasm. Spontaneous

rupture may occur with some relief of symptoms. Occasionally the clots are surrounded by edema and not readily palpable.

Thrombotic hemorrhoids usually follow one of several courses. They may be completely absorbed with only a residual asymptomatic skin tag as in the pregnant woman. In robust individuals they may become calcified and form single or multiple encysted concretions. These may be troublesome if involving the anoderm.

Occasionally they become infected forming an abscess in the perianal space. True thrombosis of the external hemorrhoidal plexus is a distinctly different pathological process which may occur with thrombosis of the internal and communicating veins. It is intravascular and usually associated with an infectious process-thrombophlebitis, phlebothrombosis or cellulitis.

The initial symptoms, usually following strained defecation, are pricking, tickling or other indefinite uneasy sensations around the anus. Depending somewhat

Fig. 1—Histologic Section of external thrombotic hemorrhoid. Note the encysting membrane.

on the size of the ensuing clot, its proximity to the anal verge and the resultant sphincter spasm, discomfort or actual pain is soon complained of and the patient becomes cognizant of a tender perianal tumefaction. Defecation is usually painful or inhibited. Symptoms gradually subside as the hematoma is absorbed and the tissue tension relaxes. Itching may be common. Occasionally the clot partially evacuates itself with a prompt relief in the intensity of the symptoms. In patients with a more or less complete perianal infiltration of shot-sized hematomata the pain is apt to be more severe and throbbing in character. An acute serous exudation sometimes supervenes in these patients and produces a highly irritating perianal dermatitis. This is not well understood and probably is a local allergic shock reaction. Intravenous clotting or thrombosis is a rare type of external thrombotic hemorrhoid and may be associated with thrombotic processes elsewhere. It is differentiated by direct palpation as a cord-like induration extending to the communicating veins.

Commonly enough these hematomata are confused with internal hemorrhoids either strangulated or merely prolapsed. Painful futile attempts at reduction are sometimes unwittingly attempted. The fixation of the hematomata is a characteristic differential point. Melanomata, condylomata acuminata, small cysts and atypical tumors may be confusing.

Hematomata of average size and involving only one side of the perianal skin usually lend themselves readily to simple evacuation which may be done as an ambulatory or office procedure. The simplest method is by evacuation of the clot or clots through an elliptical excision of the overlying skin. Squeezing out of the clot should not be done. With the single end of the scissors introduced into the proper line of cleavage between the encystment membrane and the surrounding

Fig. 2—A. External thrombotic hemorrhoid.
B. Excision of overlying skin to expose the clot.
C. Appearance of wound before enucleation (*After Yeomans*).

tissues the clot may be readily shelled out *en masse* with but little hemorrhage (Fig. 2). This simple procedure may be accomplished under local infiltration of 1 per cent novocaine combined with 10 c.c. of Anucaine or other oil-soluble anesthetic which provides immediate relief, affords the maximum of exposure and prevents postoperative pain. The wound should not be sutured or packed unless oozing is otherwise uncontrollable. To avoid recurrence of clots, bleeding should be meticulously controlled by fine absorbable suture. A simple drying powder, with a pressure dressing completes the operation. The use of thymol iodide is preferred by the author.

Complete perianal involvement of the external plexus by a shower of small shot-like hematomata appears to be an entirely different process, perhaps a local allergic shock reaction. It occurs independently but may be associated with, or

result from partial or complete strangulation of internal hemorrhoids. In the latter case there is also an actual thrombosis in the veins, and these may be removed concurrently with the internal hemorrhoids. When not definitely complicating internal hemorrhoids, they are advisedly treated conservatively with warm wet dressings, sitz baths, sedation, and dilatation. Resolution is usually rapid and the small remaining skin tags may be removed subsequently under novocaine or anucaine anesthesia. The surgical trauma and dissection required to remove a complete perianal corona of clots is frequently followed by a much longer and troublesome convalescence than anticipated and has no advantage over simple palliation.

Small isolated hematomata may be left to absorption and the pain and discomfort relieved by a simple injection of an oil soluble anesthetic, Anucaine, below and around the clot. This is a simple, safe, and very effective method which can be used anywhere.

The preparation and use of the oil soluble anesthetic requires a special technic and is described below.

Oil-Soluble Anesthetics

Since our initial experimentation and subsequent introduction of the original oil-soluble anesthetic Benacol in conjunction with Yeomans and Mathesheimer in 1927, we have had considerable further experience in its use. Despite the very occasional untoward reaction, we believe oil-soluble anesthetics to be of great value in proctologic practice. We consider Anucaine, our original modification of Benacol, as the safest of the oil anesthetics. We have used this preparation almost exclusively for the last 20 years.

The optimum benefits of the oils depend on their proper preparation and a careful technic in their injection.

It may be emphasized that the injection of oil around the anorectum is somewhat different from that of an aqueous solution, and it requires detailed anatomical knowledge of the several spaces, the nerve supply and the anorectal musculature. There are several minor but important points.

Anucaine should never be used except from ampules. Benzyl alcohol deteriorates rapidly and may become irritant. The ampules are preferably warmed before use, not boiled. The oil should be aspirated under strict asepsis into a 10 c.c. Luer Lok syringe. The injections, regardless of the location, should be made preferably through an 18 or 19 gauge needle. The needle should be kept moving as the oil is injected and the injection is preferably made only as the needle is advanced into the tissues in order to effect a layered and even distribution. Anucaine should never be injected under pressure and pooling is strictly to be avoided. If undue pressure is necessary it indicates injection into fascia or fibrosed muscle or other intolerant tissue. The principal contraindication, an infectious process in the anorectal tissues, *must be excluded.*

The untoward reactions include abscess and allergic reactions. Abscesses follow poor technic and using oil anesthetics which contain *more than* 5 per cent Benzyl Alcohol, phenol, ether, percaine, or other irritating chemicals. Further de-

tails on the indications and use of the oil-soluble anesthetics by the author are available elsewhere.

Anucaine Injection for Thrombotic Hemorrhoids

A skin wheal of 1 per cent novocaine is raised about 2 cm. from the outer margin of the hemorrhoid followed by the injection of 10 c.c. of Anucaine into the tissues of the perianal space below and on either side of it (Fig. 3). This injection is usually painless if the subcutaneous sphincter muscle is not injected. Injection of this muscle is sometimes advocated, but the author has found this to be unnecessary. After the Anucaine, additional novocaine should be injected into the anoderm just above the hemorrhoid. This is a highly sensitive area innervated from within the anal canal. This assures complete anesthesia and the clot is removed through an elliptical skin excision as described, or by any other appropriate technic.

Cutaneous Hemorrhoids

Erroneously referred to as hemorrhoids "skin tags", etc., are hypertrophic excressences of the true skin. The essential pathology is a fibrosis of the connec-

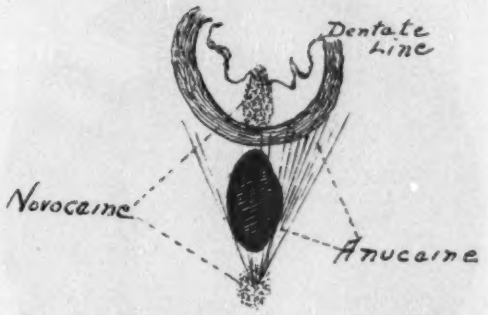


Fig. 3.—Novocaine-anucaine injection for evacuation of external thrombotic hemorrhoid. Note the anucaine is injected entirely below the clot.

tive tissue elements of the skin resulting from chronic infection, thrombosis, protracted lymph edema, and histaminia. The chronic anal fissure with its muscular spasm exemplifies this pathologic end result in the so-called sentinel pile. The chronic venous engorgement of pregnancy, anal ulcers, chronic infections, bacterial or fungus infection of the perianal skin, pruritus ani, chancroids, granulomata, etc., are common causes of skin tags. Sphincter spasm with thrombosis and poor drainage of the perianal wounds following the painless hemorrhoidectomies commonly produce residual skin tags.

Symptoms:—Skin tags are usually asymptomatic unless irritated or inflamed by local discharges, infections or true dermatological conditions. Pruritus may then be a prominent symptom.

Diagnosis:—This is usually apparent on simple inspection. Condylomata acuminata, prolapsed anal papillae, small fibromata and small external thrombotic hemorrhoids may require differentiation.

Treatment:—The treatment is either palliation or radical excision. Palliative measures include protection of the perianal skin, soothing applications of wet dressings, or ointments and regulation of bowel habits.

Surgical removal is indicated in cases in which irreversible changes have taken place in the skin. They should also be removed in the course of operative procedures on other anorectal conditions. Unless the underlying etiological factor is corrected, skin tags are prone to recurrence e.g., in pruritus ani. Removal under local procaine anesthesia combined with Anucaine affords a simple ambulant procedure.

Internal Hemorrhoids

The histopathology of internal hemorrhoids is essentially a fragmentation and attenuation of the fibroelastic stroma of the venous plexus with varying degrees of hypertrophic, atrophic and obliterative changes in the venous radicals (Fig 4).

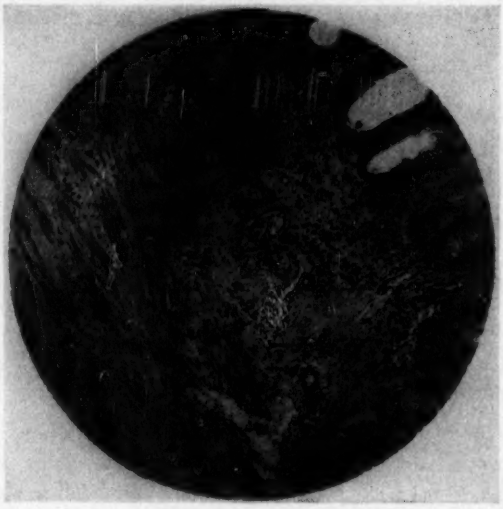


Fig. 4—Fragmentation and attenuation of the fibroelastic stroma of the internal hemorrhoidal plexus.

Cellular infiltration of the stroma with mononuclear leucocytes, plasma and particularly round cells is usually prominent and is indicative of a mild inflammatory reaction with repeated minute thrombotic, and endophlebitic processes (Fig. 5). These usually result in scarring and fibrosis both in the stroma and adventitia of the veins. The pecten tissues may be prominently involved in the fibrotic process producing a "pectenosis", anal fibrosis or pecten band.

The mucosa overlying the hemorrhoid is commonly ulcerated and the cuboidal epthelium may be replaced by squamous metaplasia or granulation tissue, which is the usual source of the bleeding.

These histopathological changes are the end result of infection, thrombosis, chronic hyperemia, tissue anoxia, sludging and histaminia in the capillary beds of the hemorrhoidal circulation as already noted. The larger capillary beds correspond to the terminal arborizations of the main hemorrhoidal arteries and this may account for the typical distribution of the larger primary hemorrhoids. These are commonly in the right anterior, right posterior and left lateral locations. Secondary hemorrhoids have a much less constant arrangement (Fig 6).

A leucocytic, plasma and round-celled infiltration is regularly observed in excised hemorrhoidal tissue as well as around the preformed ducts and anal glands. It appears that the underlying pathological factors of hemorrhoidal disease is universal and present to some degree in almost every adult.

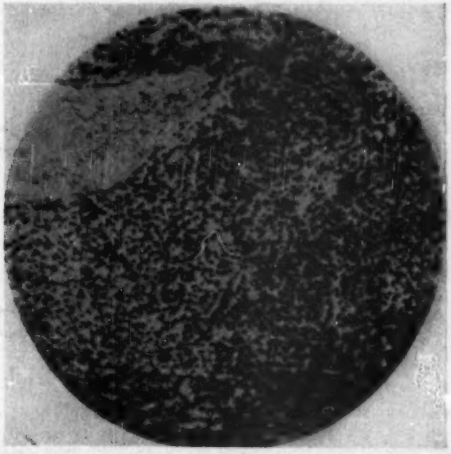


Fig. 5—Microphotograph showing round cell and leucocytic infiltration. A chronic low-grade inflammatory reaction in an apparently uninflamed hemorrhoid.

The external veins and the communicating veins gradually share in this pathological process forming the common mixed hemorrhoid. The same factors particularly histaminia are also significant in the pathology of external thrombotic hemorrhoids. Progressive increase in fragmentation and attenuation of the hemorrhoidal supports finally extend to the extensions of the conjoined longitudinal muscle, the levator ani which permits hemorrhoidal prolapse, the 2nd and 3rd degree hemorrhoids. When the extensions are pulled away from the submucus space, the pecten as well as the intermuscular septum, the hemorrhoids are usually entirely prolapsed and are not amenable to any type of injection therapy (Fig. 7).

In some individuals the supportive "*vis a tergo*" of the tissues are congenitally poor and they have carried the infant rectal pattern into adult life. Anal prolapse, entirely independent of hemorrhoids but commonly considered so, may occur on this basis. Further evidence of congenital predisposition is usually mani-

fest by the common association of a prolapsed rectum or sigmoid, herniae, enteroptosis, etc., with hemorrhoids.

Internal hemorrhoids have also been considered pathologically as vascular tumors with actual increase in their venous and interstitial components. There is little ground for this interpretation.

Thrombosed Internal Hemorrhoids

These occur in two forms, one a simple extravasation of blood in the submucosal tissues of the internal plexus and the other an actual thrombosis of the veins, usually associated with an infectious process—a thrombophlebitis or a phlebothrombosis. When active and virulent, these demand active supportive and antibiotic therapy since they may eventuate in hepatic abscesses with fatal issue.

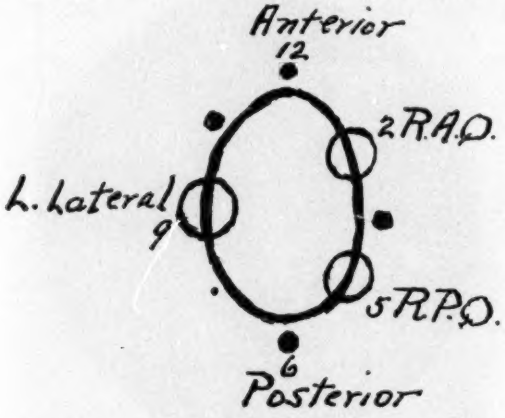


Fig. 6—Schema of the terminal arterial distribution of the superior hemorrhoidal arteries in the internal annulus. These, with their vena comites, form internal hemorrhoids as follows: Those indicated by the larger circles (the main vessels) form the primary hemorrhoids, those indicated by the dots form secondary hemorrhoids.

It may be noted in passing, that the injection therapy of hemorrhoids actually produces an aseptic thrombosis and periphlebitis which supposedly results in a "supporting" fibrosis and decreased vascularity of the hemorhoid.

Capillary Internal Hemorrhoids

These are commonly referred to as "nevoid or strawberry" hemorrhoids on account of their brighter color and punctate, granular and irregular surface which bleeds readily on contact. A true nevous of the anal canal and perianal skin is rarely observed (Fig. 8).

The criteria for the diagnosis of internal hemorrhoids vary within wide limits, sometimes tinged with overenthusiasm. It may be noted that defecation normally entails a dilatation of both hemorrhoidal plexuses with a shortening of the anal canal and perineal floor. The plexuses form a "venostatic" protection and a reflex

sensory mechanism to the anal canal for its synergic muscular contraction and relaxation, as well as the postdefecatory euphoria. It is the loss of these important anatomico-physiological mechanisms which result in the dysasthesias and dyskinesias all too commonly observed in the overenthusiastically hemorrhoidectomized patient, particularly those subjected to the Whitehead or other undesirable proctoplastic procedures.

It may also be emphasized that the symptoms all too commonly ascribed to internal hemorrhoids, particularly bleeding, partial prolapse, fullness, tenesmus, false or incomplete sense of evacuation, moisture and itching, etc., may arise from conditions entirely independent of hemorrhoids or merely associated with them. Hemorrhoidal surgery may aggravate these conditions. The proctocolitides, chronic nonspecific ulcerative colitis, mucous colitis, amebic, tuberculous, gonorrheal, the granulomata and especially new growths in the female adnexa require exclusion.

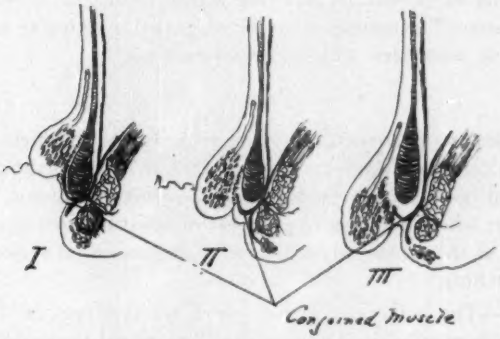


Fig. 7—First, second and third degree hemorrhoids. Schematic representation of attenuation of the supporting elements of the internal hemorrhoids, namely, the insertions of the conjoined longitudinal muscle extensions into the pecten and intermuscular septum and their continuation into the submucosa (the muculis submucosa analis). A few of these extensions reach the muscularis mucosa. Note that the intermuscular septum or groove becomes obliterated permitting progressively increasing prolapse.

Bleeding considered from hemorrhoids may arise on a normal venous plexus from simple granular erosions produced by local infectious processes, irritating chemical stools, endocrine dyscrasias, "histaminosis", menstruation, allergy, etc. Its persistence from an unsuspected malignancy following a "curative" hemorrhoidectomy is not an infrequent and embarrassing situation. The evaluation of symptoms in terms of hemorrhoidal pathology may not always be easy. Too much surgical or injection enthusiasm may be expended on symptoms rather than disease.

Internal Hemorrhoids

Bleeding and prolapse are the usual complaints. Bleeding commonly occurs during defecation (Fig. 9). It may be regular or intermittent and entirely overlooked. Its chronicity may lead to a low grade secondary anemia. That the bleed-

ing actually comes from hemorrhoids should be confirmed by anoscopic examination. That many other sources may be present even as high as the esophagus should likewise be considered.

Prolapse is not a common symptom of early hemorrhoidal disease. It usually occurs in the larger variety which have lost their supporting matrix and which may require manual replacement. Prolapse is progressive and in its early stages may be entirely unnoticed by the patient. In the so-called second degree hemorrhoids, prolapse may occur but it usually reduces itself spontaneously. A sense of incomplete evacuation may precede the complaint of actual prolapse which, if advanced, may cause a partial obstruction to complete evacuation.

Partial or repeated prolapse may result in edema congestion and thrombosis in the external plexus forming the common extero-internal or mixed hemorrhoid. Strangulation may finally occur.

Hyperactivity of the mucus secreting glands results in the excessive perianal moisture or "wetness" sometimes complained of with prolapsing hemorrhoids, and particularly those associated with anorectal prolapse.

Pruritus

"Itching piles" is a common lay expression. In our experience, however, true pruritus ani is a comparatively uncommon symptom of hemorrhoids. With chronic prolapse the anal canal may become lax and the sphincters atonic, permitting some degree of leakage which may provoke a sense of moisture, irritation and occasional pruritus. Most of the sensory dysasthesias of the anorectal region are commonly interpreted as itching.

Inspection:—The perianal skin may show old skin tags or the external component of mixed hemorrhoids. Occasionally the internal hemorrhoids are partially or completely prolapsed, as in the case of the strangulated variety in which the diagnosis is obvious. The usual advice to have the patient bear down or strain in order to extrude his hemorrhoids under pressure is usually more successful following a digital examination and anal dilatation as indicated. The local topical application of Topogesine or a local anesthesia with Procaine or Anucaine may be useful in painful conditions. Gentle assistive retraction on the part of the examiner may be helpful in revealing all the hemorrhoids, their exact location and the degree of associated rectal prolapse, all of which are significant to the treatment.

The diagnosis may also be made and confirmed by anoscopy. Situated immediately above the dentate line, which should be identified, internal hemorrhoids appear as more or less discrete swellings usually covered by a red or purplish streaked granular appearing mucosa which may bleed readily on contact. The size and extent to which the hemorrhoids prolapse may be more clearly observed when the patient strains as the anoscope is partly withdrawn and then gently reinserted. This sometimes is a painful maneuvre. Primary internal hemorrhoids are usually found in both anal quadrants on the right side and in the mid-lateral aspect of the left side.

Pathologic details of infection, ulceration, squamous overgrowth, fibrosis, thrombosis and varicose dilatation, should be noted during anoscopic observation. These are pertinent to the therapy. The type of anoscope, of which there are many, is perhaps not as important as experience in its use. However, longitudinal fenestrated anoscopes of the Brinkerhof pattern require repeated insertion and the normal prolapsing mucosa into the slot should not be exaggerated to hemorrhoidal proportions. We prefer our own revolving anoscope with a straight or slightly beveled end, a revolving handle and fixed illumination (Fig. 11).

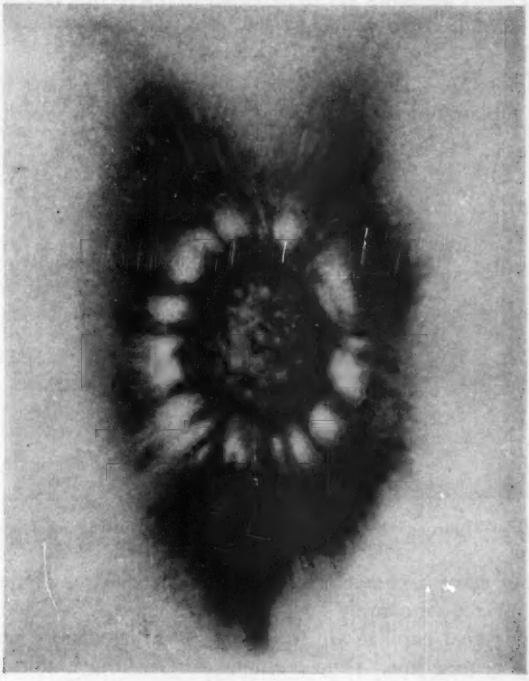


Fig. 8—The so-called strawberry internal hemorrhoid.

Digital Examination:—Hemorrhoids are usually not palpable unless the overlying mucosa is quite rugous or the hemorrhoids fibrotic. However, anal fibrosis, "pectenosis", and the pecten band which are commonly associated with internal hemorrhoids are readily palpable and significant to the surgery. Additionally the development, tonicity and contractability of the anal musculature, as well as the depth of the perineal floor are sometimes noteworthy.

Differential Diagnosis:—Ascribing the common anorectal symptoms, particularly bleeding prolapse and itching to hemorrhoids, whether these are present or

not, has been the main source of error in diagnosis, with at times disastrous results. The commoner conditions requiring exclusion include carcinoma. Cancer usually occurs above the hemorrhoidal area and is distinctly palpable as an irregular nodular firm tumefaction, more or less fixed, usually with some narrowing of the rectal lumen. Bleeding is sometimes a late symptom of cancer. Colloid carcinoma and epitheliomata, if at all suspicious, should be excluded by biopsy.

Adenomata or so-called polyps are more apt to be confused with hemorrhoids. Polyps, however, prolapse as a single tumor with a more or less discrete pedicle. Straining has little or no effect on its vascularity or color change unless it becomes strangulated. Polyps are more apt to be found in the rectum or lower sigmoid and they may be multiple. Biopsy or snare removal is confirmatory and may show malignant changes.

Villous tumors are seldom found except in adults. They are usually of much larger size and prolapse to an extent never attained by any hemorrhoids. On digital

Fig. 9—"Spurting" hemorrhage from internal prolapsing hemorrhoids.

examination they have a characteristic, loose, soft, spongy, slippery feel due to their papillary mucoid structure.

Hypertrophic anal papillae are squamous epithelial tumors which arise from the anorectal or dentate line. They are nonvascular, firm, uniformly smooth and greyish in appearance and unlike hemorrhoids may be delivered from the anal canal by digital expression.

The commonest condition confused with internal hemorrhoids is a simple anal or mucosal prolapse which may include the normal hemorrhoidal annulus in its protrusion. However the mucosal prolapse is usually entirely and uniformly annular and lacks the typical distribution and irregular globular vascular appearance of hemorrhoids. It may be noted however, that advanced hemorrhoids are usually associated with some prolapse or redundancy of the mucosa or rectal wall particularly in ptotic individuals with a shallow perineal floor. The distinction is of surgical significance. Similarly the rectocele in the female—the so-called "perineal pile"

is actually not a hemorrhoid although some enlargement of the veins are associated due to the repeated chronic congestion.

Procidentia or complete rectal prolapse has a characteristic large uniform concentric rugous appearance hardly confusable with hemorrhoids.

Finally, bleeding from the local ulcerative processes of the anal canal (fissure, etc.) and particularly from the various proctocolitides should not be unwittingly ascribed to internal hemorrhoids.

Hemorrhoidectomy in a colitis patient—particularly the chronic ulcerative type—may be followed by an intractable and complicated convalescence.

Complications of Hemorrhoids

Complications include strangulation, hemorrhage, infectious processes, thrombophlebitis and phlebothrombosis with occasional abscess.

Strangulation:—This is a fairly common complication of internal prolapsing hemorrhoids. It may involve a single hemorrhoid but more often the entire inter-

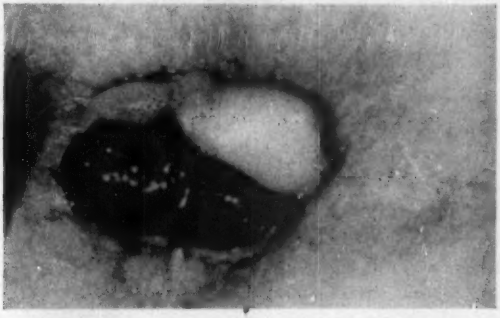


Fig. 10—Strangulated internal hemorrhoids with obstructive edema of the perianal skin.

nal hemorrhoidal annulus is involved. The extent of the pathology depends somewhat on the duration and intensity of the strangulation. The usual sequence in the pathology of strangulation was the assumption that the hemorrhoids became thrombosed and strangulated after being prolapsed and held outside the anus by the severe muscle spasm or in the grasp of the sphincters. However, it appears that the thrombosis may occur in the hemorrhoids before they are forced out. As in the case of the external thrombotic hemorrhoids the liberation of histamine or a histamine like body may be the main factor in the thrombosis and represent a local allergic tissue response. The rectal tenesmus sometimes complained of in the patient with strangulated hemorrhoids before and even after reduction is significant to a histamine etiology.

After the hemorrhoids have prolapsed, obstructive circulatory changes rapidly supervene in the external plexus and surrounding tissues (Fig. 10). Circumanal edema, with thrombosis, lymph stasis and ulceration and sloughing are the com-

mon sequelae. Pain is usually severe. A violaceous "cruller-like" perianal swelling results and is commonly mistaken for internal hemorrhoids. Unless active infection occurs, the systemic symptoms are mild. If severe, they should arouse suspicion of a virulent infection perhaps anaerobic.

Treatment:—In early cases with mild circulatory changes, a simple injection of an oil-soluble anesthetic (Anucaine) with reduction will afford immediate relief. Subsequent surgery is then elective.

In the advanced cases, the treatment is still somewhat equivocal. In general with chemo- and antibiotic therapy and careful observation, immediate removal appears to be the method of choice. In the diabetic, obese, chronic nephritic and hepatic patient, however, palliation may be the safest procedure. Local injection

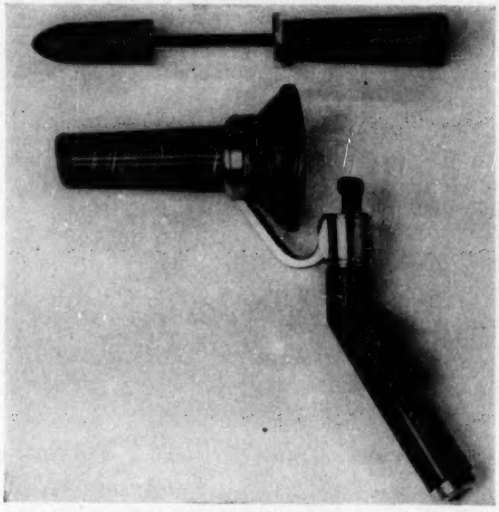


Fig. 11—Revolving anoscope with adjustable switch-light in a removable handle. Author's pattern. The shaft as well as the handle may be revolved independently as desired. (*Courtesy Miller Surgical Co., Chicago, Ill.*)

of penicillin with procaine and Anucaine with sphincter dilatation, may effect reduction and reverse the obstructive circulatory changes. If irreducible, astringent hot compresses, sitz baths and antispasmodic sedation are indicated. Rectal irrigations if properly done will avoid voluntary defecation and may prevent repetition of the prolapse.

Operative Treatment:—The distortion of the perianal tissues by the obstructive circulatory sequelae tends to obliterate the normal landmarks in the hemorrhoidal technic and it is therefore highly important to discount subsequent normal resolution. The thrombosis may have occurred in a comparatively normal internal hemorrhoidal plexus.

Simple ligation without stripping is the method of choice. When the internal plexuses are completely thrombosed, they form a more or less discrete tumor-like mass which strips up very readily from the underlying basal membrane along the internal sphincter muscle. Due to the swelling, too much anoderm and skin may be unwittingly sacrificed. Stricture following the operation for strangulated hemorrhoids is fairly common and is one of the equivocal points in favor of palliative treatment.

Prophylactic chemo- and antiobiotic therapy with alertness for complications and a careful postoperative regime are indicated, particularly in strangulation which occurs in the chronic alcoholic obese or hepatic patient.

Strangulation may occasionally complicate the injection treatment of hemorrhoids. It requires careful watching.

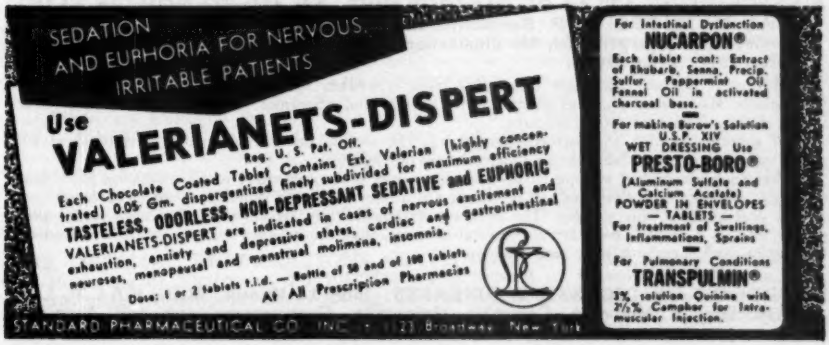
Hemorrhage—Hemorrhage of any severity rarely complicates hemorrhoidal disease. However, it may be concealed in the rectal ampulla and confuse the diagnosis until repeated bloody evacuations invite an endoscopic examination. Secondary anemia, sometimes of a severe grade, resulting from repeated bleeding, is occasionally observed. It is highly important to exclude other sources of hemorrhage which may be quite obscure.

Thrombophlebitis, Phlebothrombosis and Abscess

Strangulation with intra- or extravenous thrombosis predisposes to infectious processes and localized abscess in the submucus or perianal space may supervene. A spreading cellulitis may also occur. These sequelae usually arise in predisposed tissues as in the diabetic, nephritic, hepatic and cachectic individual. Strangulation and sloughing may also be incident to severe local trauma. Prompt active supportive and antibiotic treatment with bacteriological control is indicated. The surgery should be confined to evacuating frank abscesses or to draining, spreading uncontrolled cellulitis. X-ray therapy is sometimes of distinct value in these cases.

Thrombosis of the internal hemorrhoidal veins may occur without any external protrusion or strangulation. The interference in the venous return may result in a mild perianal edema which is usually soft without thrombosis of the external veins.

(*To be continued*)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BOOK REVIEWS

NUTRITION AND DIET IN HEALTH AND DISEASE: James S. McLester, M.D., Professor of Medicine, University of Alabama, Birmingham, Ala. Fifth Edition, 800 pages. W. B. Saunders Company, Philadelphia, Pa., 1949. Price $9.00.

This excellent text-book by an old experienced clinician has been radically and extensively revised and brought up-to-date in this fifth edition of 800 pages.

Nutrition and diet in health are intelligently and conservatively presented. The sections on nutrition in various diseases such as diabetes mellitus, and gout, digestive diseases, diseases of the cardiovascular system, diseases of the blood including pernicious anemia and hypochromic anemias; diseases of the joints, of the skin, and of the nervous system are all presented in a very readable manner, and quite instructively.

Nutrition in surgery, in industry, in endocrine disorders, in febrile diseases, in obesity and leanness, in food poisoning and allergy, and in deficiency diseases are all covered adequately and satisfactorily. Brief comments are made on diabetic coma, spontaneous hypoglycemia. In Chapter 13, consideration is given to diet and reproduction with discussions on fertility, pregnancy, and lactation.

In Chapter 19, the author discusses fully "Diseases of the kidney and urinary tract" (30 pages).

Eighty-six pages (chapters 20 and 21) are devoted to "Diseases of the digestive organs". Very helpful bibliographies follow each of the chapters.

This fifth edition of an authoritative text on nutrition and diet is highly recommended to all medical students, resident physicians and internes, general practitioners and clinicians and especially to dieticians and nurses interested in disorders of nutrition and metabolic disturbances. All medical libraries should have readily available copies of this book.

ELEMENTS OF MEDICAL MYCOLOGY: Jacob Hyams Swartz, M.D., Assistant Professor of Dermatology, Harvard Medical School and Postgraduate Medical School; Dermatologist, Massachusetts General Hospital, in collaboration with Ethel M. Rockwood, M.D., Dermatologist, Massachusetts General Hospital. Introduction by Fred D. Weidman, M.D., Professor of Dermatological Research, University of Pennsylvania. Second edition. 240 pages. Grune and Stratton, New York, 1949. Price $5.50.

This monograph on mycology is based chiefly on the author's own experience. The author, who is a dermatologist and well trained in the field of mycology, has prepared an excellent condensed guide on medical mycology, which will be very helpful to the students of mycology and dermatology and will be quite useful to all practicing physicians, clinicians, and surgeons, who have an interest in conditions caused by fungi. The author divides the book into nine chapters and a closing "Glossary" and index.

Following a general discussion is a chapter on Diagnosis of Fungus Infection. Then follow chapters on Blastomycetes; Yeastlike Fungi; Genus Microsporum (Gruby 1843); Genus Trichophyton; and other pathogenic fungi, with helpful bibliographies. "Common contaminants and probable pathogens" and immune reactions are briefly, but satisfactory discussed. Antibiotics and newer chemotherapeutic agents—including sulfanomides, penicillin, and streptomycin are very briefly mentioned, as they "have proved to be of little or no value in the treatment of superficial mycoses".

This monograph is recommended as a useful small guide on the subject of medical mycology.

A DESCRIPTIVE ATLAS OF RADIOGRAPHS. AN AID TO MODERN CLINICAL METHODS: A. P. Bertwistle, M.B., Ch. B., F.R.C.S.Ed. Seventh Edition, revised and enlarged with 980 illustrations. 622 pages. C. V. Mosby Co., St. Louis, Mo., 1949. Price $16.00.

This most unusual and most interesting book is written by a clinician for clinicians. In this fine volume the author portrays many of the normal and abnormal conditions that are met with in the daily practice of most clinicians.

Of especial interest to gastroenterologists, is the section on the "Alimentary System" of 111 pages, containing many informative illustrations—188 figures.

This finely illustrated volume on good quality paper is recommended to all practicing physicians, residents, students and clinicians, all of whom are interested in the radiologic aspects of many conditions portrayed in this volume. The reviewer enthusiastically recommends this book to all physicians, and as a "must" book for all hospital libraries, that the internes and senior medical students and junior staff members, may read and learn.

HANDBOOK OF DIGESTIVE DISEASES: John L. Kantor, M.D., F.A.C.P. Late Associate in Medicine, Columbia University, Gastroenterologist and Associate Roentgenologist, Montefiore Hospital, New York, and Anthony M. Kasich, M.D.,

F.A.C.P. Lecturer in Medicine, Columbia University; Adjunct Physician Montefiore Hospital, Assistant Visiting Physician, Bellevue Hospital, Assistant Adjunct Gastroenterologist, Lenox Hill Hospital, New York. Second Edition. 658 pages. Illustrated. C. V. Mosby Company, St. Louis, Mo., 1949. Price $11.00.

This new compact volume on digestive diseases will be favorably received by all general practitioners of medicine, medical students, internes, resident physicians, and young gastrenterologists.

The physician who is developing an interest in gastroenterology will gain much valuable information from this volume, by two experienced gastroenterologists. It is unfortunate that the senior author, the late Dr. John L. Kantor, did not live to see the completed work by his close associate Dr. Anthony M. Kasich. Dr. Kasich has attempted to include many of the essential facts concerning digestive diseases. The author, probably, will write a more extensive and more inclusive textbook on gastrointestinal diseases during the next five years, which should be of great value to the more experienced clinicians and gastroenterologists.

In the meantime, the reviewer recommends this concise presentation of the essentials of gastrointestinal diseases, to all those interested in this branch of internal medicine. This is truly the best small volume available, which covers the subject so well, a real practical "handbook".

P-Q-R-S-T A GUIDE TO ELECTROCARDIOGRAM INTERPRETATION: Joseph E. F. Riseman, M.D., Associate in Medicine, Harvard Medical School; Instructor in Medicine, Tufts Medical School; Visiting Physician, Beth Israel Hospital, Boston, Mass. Second Edition. 84 pages. The MacMillan Company, New York, 1949. Price $3.50.

This is an excellent little guide for the young physician and beginner interested in the basic fundamentals of electrocardiography. In this little "booklet", the senior medical student, the interne and resident, and the practitioner will find instructions for the correct interpretation of the more common abnormalities met with in electrocardiographic tracings.

This booklet contains, clearly and quite informatively, the technic of taking tracings and the method of studying electrocardiograms, and the normal variations in standard and precordial leads.

The author presents in very readily understandable manner the abnormalities encountered of the P waves, P-R intervals PS-T segments, precordial lead IV, the various ventricular patterns, and the multiple chest leads (C or V). The author includes 31 short case histories with illustrative electrocardiograms.

This little work is highly recommended for senior medical students, and all young physicians and practitioners interested in the interpretation of the more common abnormalities met with in the study of electrocardiograms.

TEXTBOOK OF VIROLOGY: A. J. Rhodes, M.D., F.R.C.P. (Edinburgh) Connaught Medical Research Lab. and School of Hygiene, University of Toronto, and C. E. Van Rooyen, M.D., D.Sc., M.R.C.P., Connaught Medical Research Lab. and School of Hygiene, University of Toronto. 312 pages. Thomas Nelson and Sons, New York, Toronto, 1949. Price $5.00.

This compact volume written by two noted experts, who also wrote the large volume on "Virus Diseases of Man", second edition 1948, will indeed be very helpful and most informative to all students of the subject, and to all medical practitioners. This work gives a succinct account of the essential features of the virus and rickettsial diseases of man. This book should also be helpful to veterinary students and veterinarians.

Among the topics briefly but quite adequately discussed are: The economic importance of virus and rickettsial infections in man and animals; classification of viruses and rickettsiae; properties of viruses, technical methods and apparatus in virus research; immunity mechanisms interference phenomenon; prospects in treatment of virus diseases; a spread of virus diseases; some skin diseases; small pox, measles, mumps, colds, influenza, eye diseases, yellow fever, poliomyelitis, rabies, encephalitis, and biology of the Rickettsiae pathogenic to man; "Infectious Hepatitis" and "Homologous Serum Jaundice" are instructively and concisely considered in chapters 19 and 20 (7 pages). There is also a brief discussion of "Laboratory Methods in the Diagnosis of Neurotropic Virus Diseases" (3½ pages).

This little volume is recommended to students of medicine and veterinary medicine and to general practitioners. For wider study one may read the large volume on "Virus Diseases of Man".

THE OFFICIAL PREPARATIONS OF PHARMACY: Charles Oren Lee, Ph.D., Professor of Pharmacy, Purdue University School of Pharmacy, Lafayette, Ind. 528 pages. Illustrated. C. V. Mosby Co., St. Louis, Mo., 1949. Price $5.50.

This volume contains understandable and informative discussions of the problems pertaining to the manufacture of pharmaceutical preparations which are official in the Unites States Pharma-

copeia 13 and the National Formularly 8. This compact volume contains much helpful information for students of pharmacy and for the medical students interested in the preparation of many of the official pharmaceutical remedies, solutions, infusions, decoctions, mixtures, and magmas, mucilages, gels, and jellies, syrups, juices, ointments, extracts, liniments, powders, cachets, douches and capsules, etc.

This book is recommended for students in the early period of their professional studies.

ELECTROCARDIOGRAPHIC TECHNIQUE: A MANUAL FOR PHYSICIANS NURSES AND TECHNICIANS: Kurt Schnitzer, R.T., M.D. 96 pages. Grune and Stratton, New York, 1949. Price $3.50.

This booklet deals exclusively with electrocardiographic technic, and will be helpful to young physicians, medical students, technicians, and practitioners who are owners of electrocardiograph machines, and occasionally wish to use them.

Technicians will appreciate the simple clear-cut explanation and discussions presented by the author.

Students, technicians, and young practitioners will do well to study this booklet carefully.

This work by Dr. Schnitzer, the work on "Electrocardiogram" by Dr Sigler (Grune and Stratton) and Dr. Riseman's guide "P-Q-R-S-T" (MacMillan Company) should be studied by all students, internes, residents, and practitioners who are at all interested in electrocardiography.

Dr. Schnitzer's booklet is recommended to all those interested in correct electrocardiographic technic.

ARTERIAL HYPERTENSION, ITS DIAGNOSIS AND TREATMENT: Irvine H. Page, M.D., and Arthur Curtis Corcoran, M.D., Research Division of the Cleveland Clinic Foundation, Cleveland. Ohio. 400 pages. The Year Book Publishers, Inc., Chicago, Ill., 1949. Price $5.75.

This compact and useful book on hypertension will aid all readers and students of the subject, resulting in clearer understanding of the etiology and management of this often troublesome problem. The various forms of hypertension are adequately discussed: congestive heart failure, renal failure, hypertension and pregnancy, and various methods of treatment are clearly presented and expertly discussed.

The volume is enthusiastically recommended to practitioner and student, to clinician and specialist, and to all internes and residents.

1949 YEARBOOK IN DRUG THERAPY: Edited by Harry Beckman, M.D., Director, Department of Pharmacology, Marquette University School of Medicine. 718 pages. The Year Book Publishers, Inc., Chicago, Ill. Price $4.75.

Allergy, cardiovascular diseases, dermatology and infectious diseases, deficiency diseases, neoplastic diseases, neuropsychiatry, ophthalmology, venereal diseases and various surgical specialties all receive consideration in this compact little volume of 718 pages. Drug therapy comes into its own and reaches high places in this work! This book is recommended for busy general practitioners, and medical students, and nurses, as well as for internes and residents in hospitals and clinics.

TEXTBOOK OF BACTERIOLOGY. Eleventh Edition of Muir and Ritchie's "Manual": C. H. Browning, M.D., LL.D., D.P.H., F.R.S., Gardiner Professor of Bacteriology, University of Glasgow, and T. J. Mackie, C.B.E., M.D., L.L.D., D.P.H., Professor of Bacteriology, University of Edinburgh. 907 pages. Oxford University Press, New York, N. Y., 1949. Price $12.75.

The first edition of this work appeared 52 years ago. This is a new work by the present authors—although considered also as the eleventh edition of "Muir and Ritchie's Manual". The whole book has been recast, and most of the reading matter has been rearranged and revised and brought up-to-date. Many improvements are noted since the tenth edition 11 years ago. The book is rich in the basic information given by laboratory and clinical investigations.

Microorganisms responsible for diseases in man, as well as the infections of animals receive ample consideration by the experienced authors.

The authors retain, for the most part, the nomenclature of previous editions of Muir and Ritchie's Manual. Among the subjects informatively presented are the morphology and physiology of bacteria, the relation of bacteria to disease, immunity, staphylococci and streptococci, the pneumococcus, meningococcus, gonococcus, diphtheria bacillus, tubercle bacillus and saprophytic acid-fast bacilli, leprosy bacillus, the coliform bacilli, the brucella group, viruses, and fungi, etc., and malaria plasmodia, and pathogenic protozoa—trypanosmes, leishmania, toxoplasma, chemotherpy (Chapter 37), the bacteriology of air, water, sewage, soil, and milk from the hygienic standpoint are also adequately and satisfactorily discussed.

An appendix on culture, methods, microscopic methods, serological methods, and methods of demonstrating filterable viruses is a valuable and helpful addition.

An excellent bibliography of 54 pages includes the more important references to the literature.

This work is highly recommended as a most authoritative and reliable source of information for the student, bacteriologist, and investigator. It is a most welcome addition to every laboratory and research library. All hospital laboratories and all public health laboratories should have a copy of this book, for ready reference.

PHYSIOLOGY OF THE NERVOUS SYSTEM: John Farquhar Fulton, D.B.E., M.D., D.Sc., LL.D., Sterling Professor of Physiology, Yale University, Formerly Fellow of Magdalen College, Oxford. Third Edition, Revised. 667 pages. Oxford University Press. 1949. Price $10.00.

This revision of an excellent work on neurophysiology brings the text up-to-date. The recent advances in neurosurgery, particularly the use of frontal lobotomy for the relief of abnormal mental states, and the surgical procedures for the relief of pain in incurable diseases, make the study of the physiology of the nervous system an important one for student, neurologist and surgeon—as well as clinician. Dr. Fulton has covered the subject most thoroughly in a very readable and interesting manner. All of the 26 chapters are well done, and the various historical notes add very interesting information.

The excellent bibliography and author index of 72 pages of references, should be most helpful to the investigator and student. This volume is authoritative and is the best one volume physiology of the nervous system available for all those persons interested in this subject.

The book is recommended to students, clinicians, neurologists, and neurosurgeons and to teachers of physiology.

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Relationship of Stress to Autonomic Lability

Studies in psychosomatics have shown that functional disorders often are a result of the patient's inability to adjust to emotionally stressful situations (stressor factors).

Nervous tension and chronic anxiety, discharged through a labile Autonomic Nervous System, can cause somatic disturbance. [1,2] Such states may involve any one of the organ systems or several at one time. [1,3] The outline below relates gastrointestinal and cardiovascular symptomatology to the exaggerated response of the autonomic nervous system.

	Physiologic Effects of Autonomic Discharge	
	Sympathetic	**Parasympathetic**
Gastro-intestinal	Hypomotility Intestinal Atony Hyposecretion Reduced salivation	Hypermotility Gastrointestinal spasm Hypersecretion
Cardio-vascular	Rapid heart rate Peripheral vaso-constriction	Slow heart rate Vasodilatation
Functional Manifesta-tions	Palpitation Tachycardia Elevated B. P. Dry mouth—throat	Heartburn Nausea-vomiting Low B. P. Colonic spasm

Data here tabulated is from references [3,4,5,6,7] given below.

Diagnosis of functional disorder is supported by the following indications of autonomic lability:

Variable Blood Pressure
Body Temperature Variations
Changing pulse rate
Deviations in B. M. R.
Exaggerated Cold Pressure Reflex
Glucose Tolerance Alterations

Therapy in these cases is directed toward: 1) relief of symptoms by drug therapy (so making the patient more amenable to psychotherapy); 2) psychotherapeutic guidance in making adjustment to stressful situations and correction of unhealthy attitudes.

Clinicians who have studied these disorders, including those of the menopause, report that good therapeutic results are produced by combined adrenergic (ergotamine) and cholerinergic blockade (Bellafoline) with central sedation (phenobarbital) [8,9,10]. A convenient preparation of this nature is available in the form of **Bellergal Tablets.** Functional disorders are long-term therapeutic problems; therefore, drug treatment by the following method is recommended: 5 or 6 tabs. per day for the 1st week; then gradually reduce to the smallest dose effective in maintaining the patient symptom free (average: 3 tabs. daily). Interrupt for 1 week out of every month to assess results.

1. Ebaugh, F.: Postgrad. Med. *4:* 208, 1948. 2. Wilbur, D.: J.A.M.A. *141:* 1199, 1949. 3. Williams, E. and Carmichael, C.: J. Nat'l. Med. Assoc. *42:* 32, 1950. 4. Goodman, L. and Gilman, A.: The Pharmacological Basis of Therapeutics, The Macmillan Co., 1941. 5. Katz, L. et al: Ann. Int. Med. *27:* 261, 1947. 6. Weiss, E. et al: Am. J. Psychiat. *107:* 264, 1950. 7. Alvarez, W.: Chicago Med. Soc. Bulletin, 581, 1950. 8. Rakoff, A.: A Course in Practical Therapeutics, Williams and Wilkins, 1948. 9. Karnosh, L. and Zucker, E.: A Handbook of Psychiatry, C. V. Mosby Co., 1945. 10. Harris, L.: Canad. M.A.J. *58:* 251, 1948.

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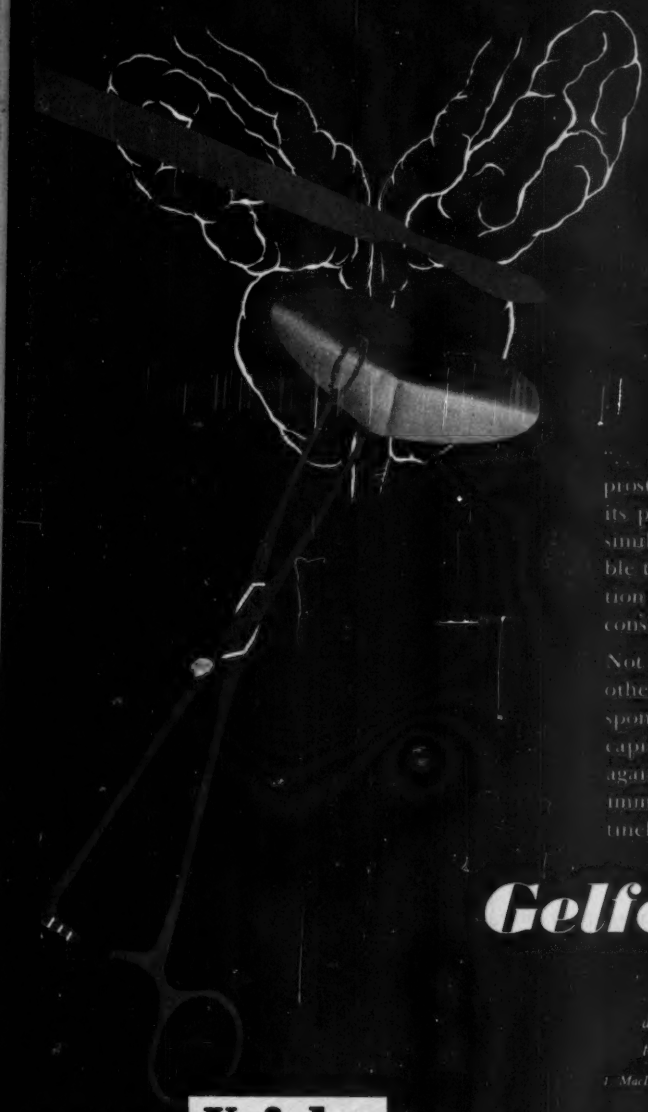

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1. MacDonald, S. A. and Powell, R. E.: J. Urol. 57: 812, 1947.

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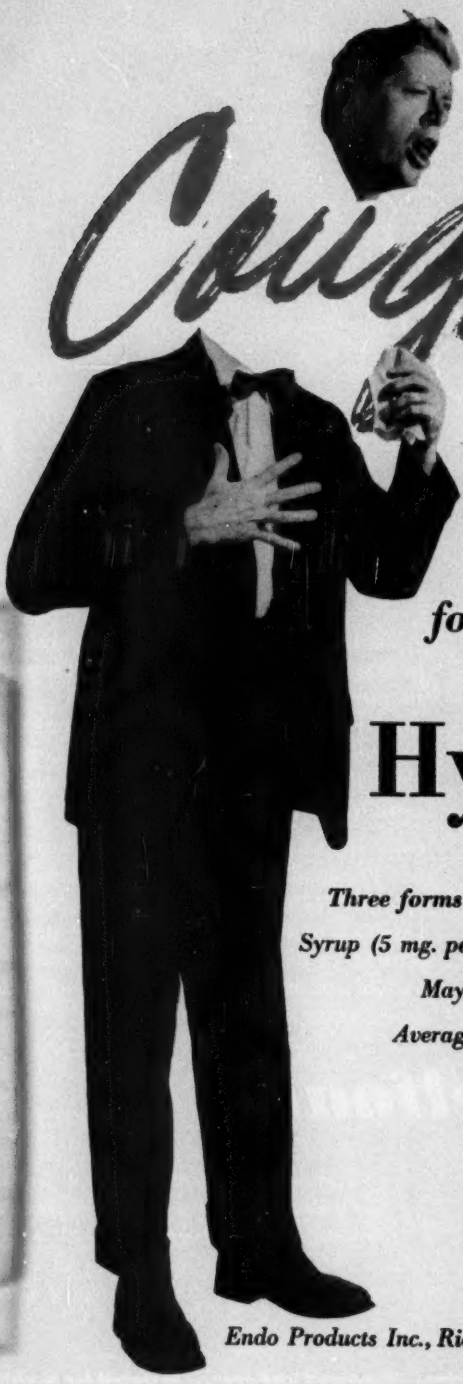

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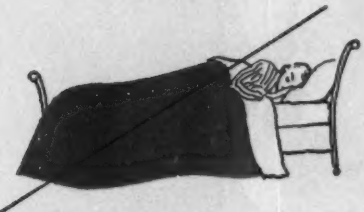

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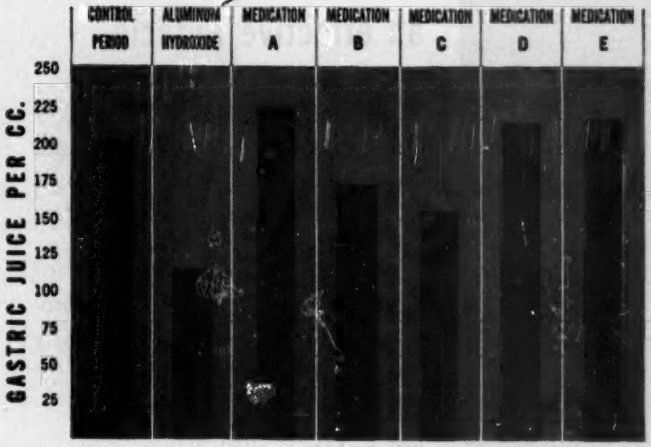

Average volume of gastric secretion from midnight to 5:30 A.M. in patients with duodenal ulcer treated with various medications *during the day only*. Aluminum hydroxide depresses volume of gastric secretion more effectively throughout entire night period. (Adapted from Brehaus, H. C., Akre, O. H., and Eyerly, J. B.: *Gastroenterology*, 16:172, Sept., 1950.)

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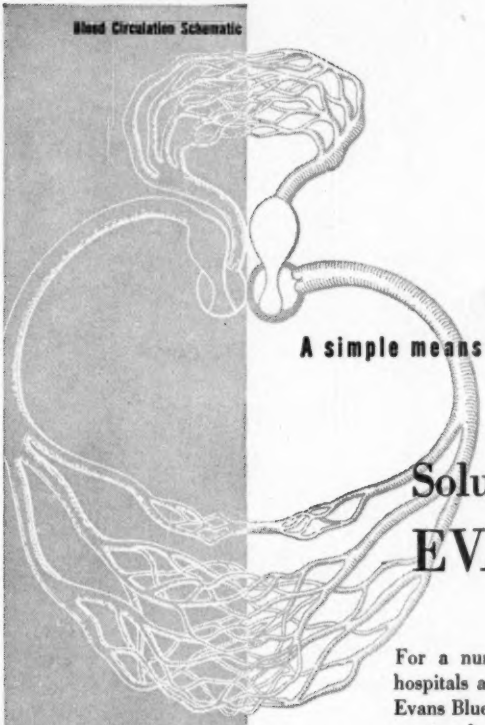

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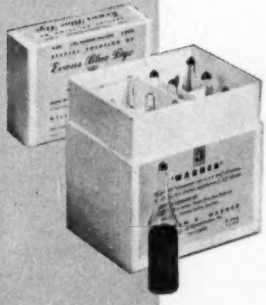

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1. Larimore, J. W.: Southern M. J. *44*:742, 1951.

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